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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

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Ann Arbor, Michigan

1958 Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

November 6, 7, 8, 1958

Dallas, Texas

SAMUEL CHARLES MILLER

November 5, 1903 February 8, 1958

Dr. Samuel Charles Miller, widely known and respected Professor and Chairman of the Department of Periodontia and Oral Medicine of New York University College of Dentistry, passed away in New York on February 8, 1958. Dr. Miller's contributions to periodontology are indeed noteworthy and since he earned for himself a host of personal friends, his death is a great loss.

Dr. Samuel Charles Miller was active in the American Academy of Periodontology and the Academy of Dental Medicine, as well as various other dental organizations. He was author of three textbooks and innumerable articles, including some in Spanish, and was a popular lecturer. His keen understanding of the clinical aspects of periodontology, his facility of speech and ready wit, made him a very popular and effective teacher; he supervised the education of over 500 postgraduate students from 43 states and 41 foreign countries.

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY-EIGHT

Effect of C-Avitaminosis on the Supporting Structures of the Teeth

by JENS WAERHAUG, D.D.S., PH.D.,[1] *Oslo, Norway*

THE main feature of scurvy is, according to Wolbach and Howe,[12] the inability of the fibroblasts to form intercellular substances. This is corroborated by Penney and Balfour[6] who states that the failure of wound repair in depleted animals is associated with the failure of formation of extracellular material in both homogenous and fibrous form. Robertson and Schwartz[7] observed that the rapid formation of large amounts of collagen containing tissue increased the nutritional ascorbic acid requirement in guinea pigs.

Hemorrhages of the gum and loosening of the teeth have for centuries been recognized as clinical symptoms of scurvy. Extensive histo-pathologic studies on scurvy in man have been carried out by Westin.[10] His clinical examination revealed severe hemorrhages, destruction of the epithelium and increased pocket depth as the result of degeneration of the blood vessels and capillary stasis. The increased mobility of the teeth was considered to be caused mainly by the scorbutic destruction of the supporting structures. When vitamin C was added to the diet, a dramatic healing of the periodontal structures was observed, and he maintains that a healed scurvy hardly leaves any permanent damage. Similar clinical and histologic observations have also been reported by Orban and coworkers.[5] Crandon,[2] who placed himself on a diet free from vitamin C for 6 months, found that the changes in the supporting structures of the teeth appeared late in comparison to alterations occurring in skin, as well as impaired wound healing and petechia. Only a slight bogginess of the gum and an interruption of the lamina dura in the X-ray picture could be observed. No bleeding occurred on brushing and a biopsy revealed normal tissue.

The effect of vitamin C deficiency on the periodontal structures has also been studied on guinea pigs,[1,3,9] monkeys[4,8] and even in rats.[11] The findings have been similar to those made in man.

As vitamin C deficiency is generally accepted as one of the predisposing factors in periodontal disease, the present study was started with the purpose of trying to find out whether a longstanding vitamin C deficiency in monkey would lead to a clinical and histopathologic condition similar to that of pyorrhea in man.

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Acknowledgment. The experiment on which this article is based was undertaken at the University of Minnesota, School of Dentistry, Minneapolis 14, Minn., U.S.A. The author likes to express his sincere thanks to Dr. W. Crawford, dean of the Dental School and Dr. H. A. Zander, chairman of the division of Periodontology, for excellent collaboration.

MATERIAL AND METHOD

Four young monkeys were used for the experiments. Their exact age was unknown, but it should be noted that only the permanent central incisors and first molars had broken through. The animals were individually housed and fed vitamin C free diet consisting of dried whole milk mixed with water in the ratio 1:9. This mixture was boiled in a copper kettle for half an hour under constant insufflation of compressed air to destroy any trace of ascorbic acid in the dried milk.

The milk was then allowed to cool, whereupon cod liver oil and brewers yeast were added to provide an ample supply of vitamin A, B and D. The animals were maintained exclusively on this diet along with water ad libitum until grave scurvy was observed. Then small amounts of orange juice were added to the diet, which prevented the animals from dying without, however, changing the level of scurvy. In order to evaluate the effect of oral hygiene, the teeth on the left side were brushed daily with a very soft brush while those on the right side were not. After removal, the jaws were fixed in 10% formalin, decalcified in 5% nitric acid and embedded in celloidin. The sections were stained with hematoxylin and eosin and Mallory's connective tissue stain.

OBSERVATIONS

Two of the animals died after 42 and 75 days respectively, without exhibiting typical clinical signs of scurvy. Obvious clinical signs of scurvy appeared after about 90 days in the remaining two animals, who lived for 234 and 257 days respectively. After 120 days increased mobility of the teeth were observed and the mobility progressed steadily. At the end of the experiment the teeth were so loose that the animals could not chew slices of apples, 2 mm. thick. Concomitant with the loosening of the teeth, the gingivae became flabby and could be blown away from the tooth surface with a gentle jet of air. Bleeding during brushing of the teeth increased after 120 days and only the softest brushes could be used. At the end of the experimental period, rather large accumulations of calculus were found to be present on the unbrushed side, but not on the buccal and lingual surfaces of the brushed side. There was obvious gingivitis in the area corresponding to the calculus. On the brushed side the appearance of the gingivae was conspicuously better, although bleeding was profuse even here.

During life the gingivae reached up to the supragingival calculus, whereas a certain distance (up to 1 mm) was observed after the jaws had been taken out, indicating a post-mortem "recession" of the gingival margin.

Although clinical signs of scurvy were not observed after 75 days, the histologic examination of the jaws and teeth revealed disturbed bone formation and increased osteoclastic resorption. After 234 and 275 days the histologic changes were much more advanced, and sections from monkey 10 (275 days) will be used to illustrate these changes. In this animal the four primary lateral incisors were extracted two weeks after the initiation of the experiment and two primary molars were removed two months later. The four permanent lateral incisors and one premolar as well as the four permanent second molars broke through during the experimental period. Ten primary teeth remained in their proper places throughout the entire experimental period.

Dentin. The odontoblasts in the erupting teeth were dedifferentiated although some dentin formation occurred in the apical part (Fig. 1, bottom right). The pulp tissue showed some degeneration and rather few vessels.

Bone. Bone formation was arrested. An organized layer of osteoblasts and osteoid tissue was not found anywhere (Figs. 1-7). The newly formed bone revealed the typical scorbutic characteristics: thin trabeculae and small marrow spaces. Osteoclastic resorption was a common finding, and in many areas extensive resorptions of the alveolar wall had taken place (Fig. 5).

Connective tissue. The collagen was nearly completely broken down and only scattered "islands" could be observed in the Mallory stained sections (Figs. 3-6). Fibers transversing the periodontal membrane from the cementum to the alveolar wall did not exist. Sharpey's fibers were in no case

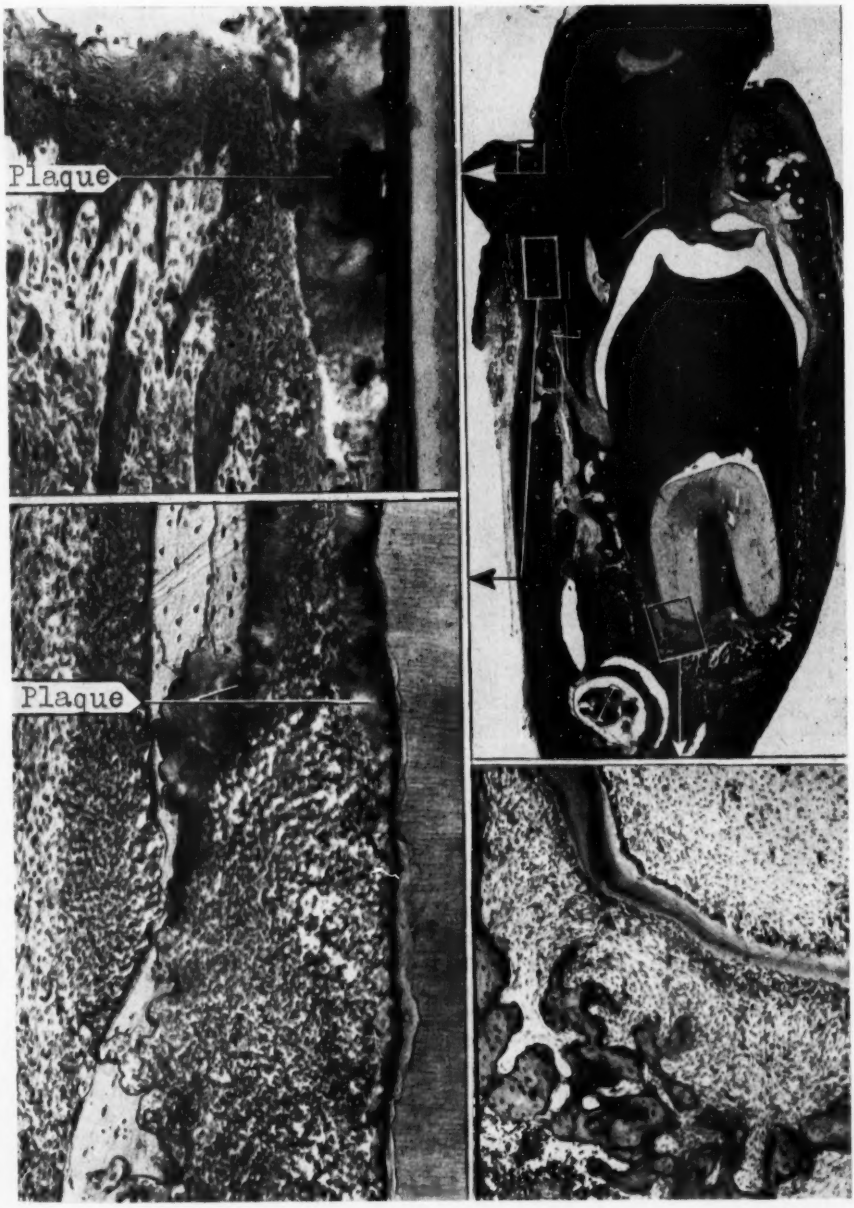


Fig. 1. Monkey 10. Lower second primary molar with underlying second permanent premolar (hematoxylin eosin). **Permanent premolar:** Enamel apparently normally developed. Dentin-formation in apical area retarded and odontoblasts dedifferentiated (bottom right). Newly formed bone of scorbutic type with thin trabeculae and narrow marrow spaces. **Primary molar:** Resorption of root. Width of periodontal membrane on buccal side approximately normal. Formation of subgingival bacterial plaque and thickening of epithelium (top left). Plaque formation extending between necrotic alveolar bone and tooth below epithelial cover, and pus formation (bottom left).

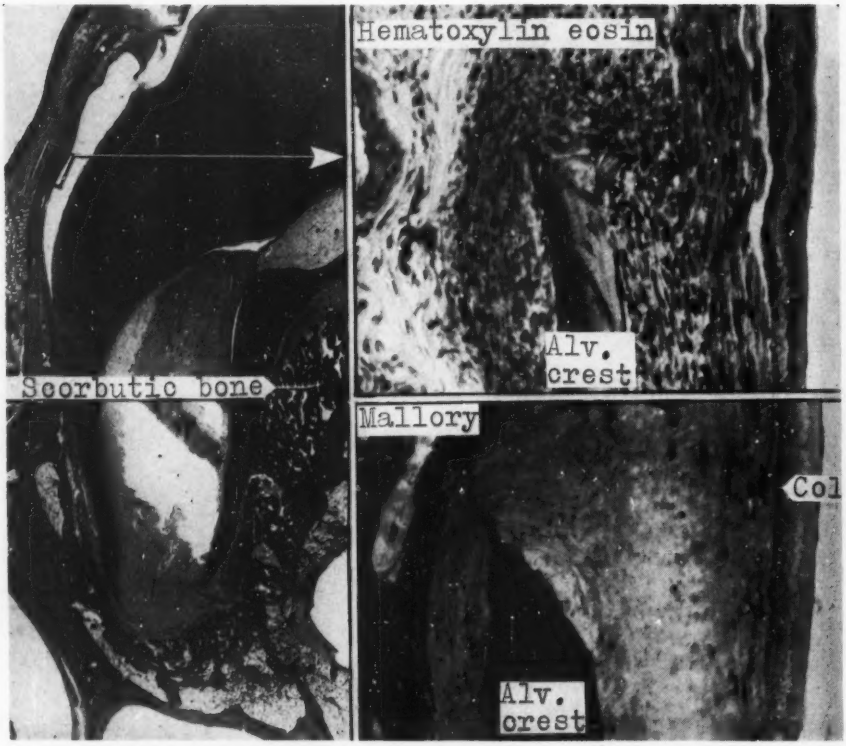


Fig. 2. Erupting upper right second molar (hematoxylin eosin and Mallory). Ameloblast of approximately normal appearance for that stage of eruption at level of alveolar crest (top right). Sections from same area stained in connective tissue stain revealed nearly complete destruction of collagen (bottom right). Excepted are basement membrane (Col) and area outside alveolar crest. Normal width of periodontal membrane.

seen to extend from the alveolar wall (Figs. 4 and 6) but were regularly seen to extend from the cementum (Figs. 3-6). They were best developed immediately below the epithelial cuff (Figs. 3-6) and decreased in length and numbers towards the apex where in some places they were barely visible (Figs. 4, bottom left, and 5, bottom right). Epithelial islands were present in normal form and numbers even in areas where the destruction of the Sharpey's fibers were most advanced (Figs. 3-6). Hemorrhages of different sizes were found regularly in the periodontal membrane, the gingivae and the bone (Fig. 5).

Epithelium. The amelogenesis did not seem to be affected in developing teeth (Fig. 1), and ameloblasts, which appeared normal for that stage, were seen in erupting teeth at the level of the alveolar crest (Fig. 2, top right) in spite of the fact that the collagen was almost completely broken down in the same region (Fig. 2, bottom right). In the fully erupted teeth some degeneration of the cells of the epithelial cuff had as a rule occurred although the overall picture could be close to normal (Figs. 8 and 9). In many cases the cells were found in close proximity to a cuticle on the surface of the enamel all the way to the gingival margin. In some cases the epithelial cuff was very narrow (Figs. 4 and 6) or entirely lacking (Fig. 5). The epithelium, where it bordered directly upon plaque, was degenerated and infiltrated with

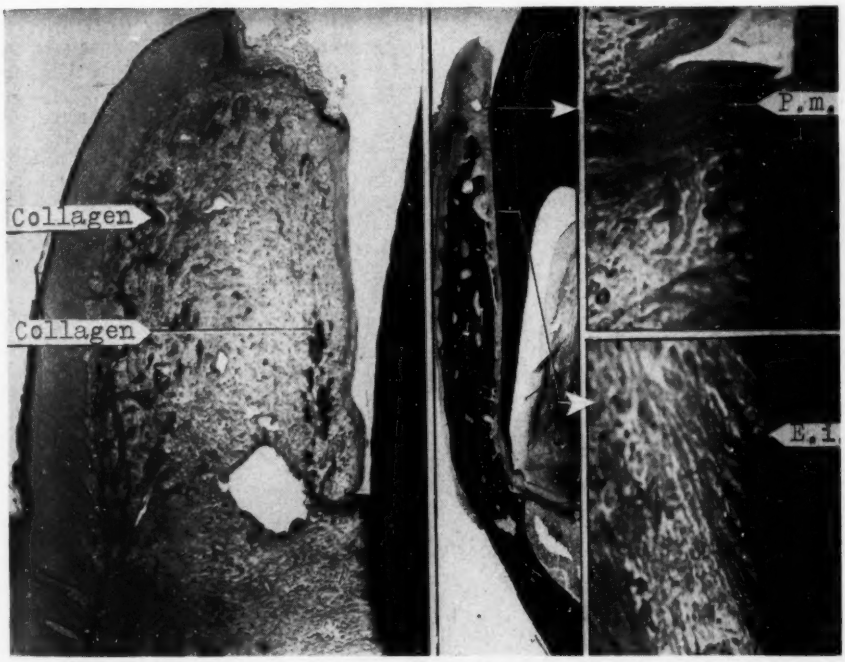


Fig. 3. Fully erupted lower right second molar (Mallory). Periodontal membrane widened. Alveolar crest of normal height. Collagen nearly completely broken down except for scattered "island" (left). Well maintained periodontal membrane fibers (P.m.) just below cemento-enamel junction (top right). Collagen fibers adjacent to cementum preserved while destroyed in rest of periodontal membrane (bottom right). E.i. epithelial islands.

polymorphonuclear leukocytes (Fig. 1); sometimes it could be completely lacking (Fig. 7, left). In the majority of cases no downgrowth of the epithelial cuff below the cemento-enamel junction had occurred (Figs. 2, 3, 5, 7, 8 and 9). The deepest epithelial cells were always seen to end at the upper periodontal fibers attached to the cementum (Figs. 3-6). In a few cases varying degrees of epithelial downgrowth had taken place (Figs. 1, 4 and 6).

Plaque formation. Formation of subgingival bacterial plaque was found in some cases (Figs. 1 and 7) and in one case the plaque had even covered the inside of the alveolar wall, which was necrotic (Fig. 1). Where the soft tissue was found in contact with the plaque, an inflammatory reaction was obvious.

In the sections from monkey 10 the width of the periodontal membrane and the downgrowth of epithelium below the cemento-enamel junction were measured. These measurements revealed that the periodontal membrane was conspicuously wider around the permanent teeth (average 0.42 mm) than around the primary teeth (average 0.26 mm). The downgrowth of epithelium below the cemento-enamel junction varied between 0.3 and 1.5 mm on the primary teeth and 0.0 and 2.0 mm on the permanent teeth, being zero in 24 out of 28 measured surfaces of the latter group. There was no conspicuous difference in the downgrowth on the brushed and unbrushed teeth, but the inflammation was on an average slightly less pronounced on the brushed side.

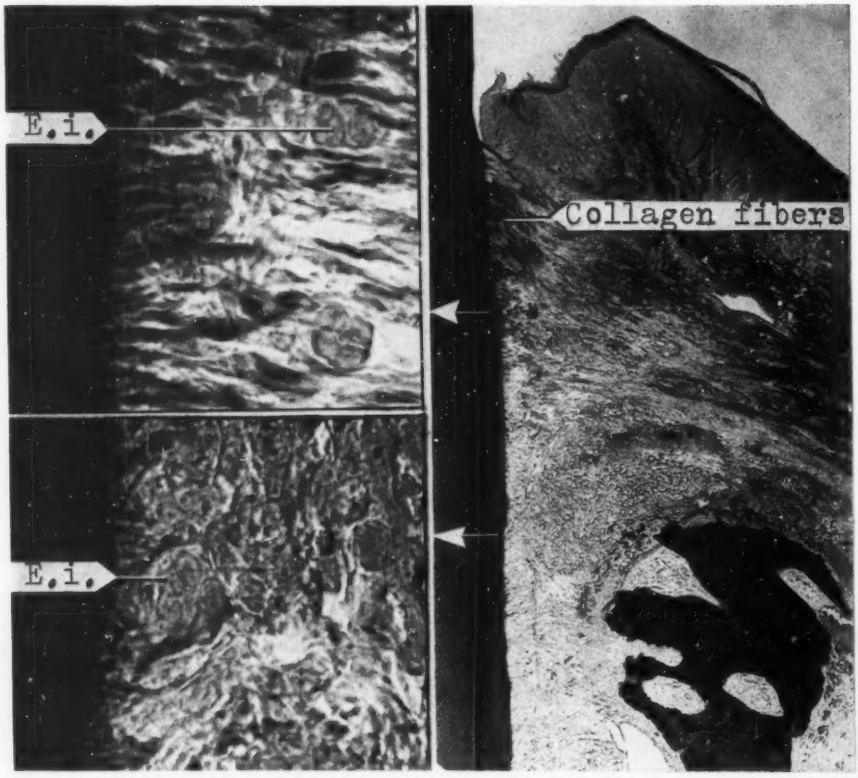


Fig. 4. Lower right primary cuspid (Mallory). Periodontal membrane widened. Collagen nearly completely broken down except for principal fibers just below deepest point of epithelial cuff. Approximately normal epithelial island (E.i.) between collagen fibers (top left). Degenerated epithelial island (E.i.) outside scattered remnants of collagen fibers adjacent to cementum (bottom left).

DISCUSSION

This series demonstrates clearly that the supporting structures are severly affected in vitamin C deficiency. The main features were the nearly complete destruction of the collageneous fibers, formation of an inferior type of bone and dentin, bone resorption and capillary extravasation. These are all recognized symptoms of scurvy. The destruction of the periodontal fibers explains the initial increased mobility of the teeth, and the resorption of the alveolar wall explains the extreme mobility during the later stages. It was rather surprising that downgrowth of epithelium below the cemento-enamel junction did not occur faster and more frequently in spite of the severe destruction of the periodontal fibers. However, it should be noted that the principal fibers just below the epithelial cuff were always those least affected by the C-avitaminosis. Thus, the anatomy of the supporting structures is well suited to prevent permanent damage in the form of epithelial downgrowth and pocket deepening.

This series corroborates the observations made by Glickman[3] that the destruction of the periodontal fibers is most advanced on the alveolar wall side. In none of the sections from monkey 10 were Sharpey's fibers seen to extend from the alveolar wall

while some Sharpey's fibers were always found on the cementum. There must be a reason for this discrepancy, and it seems natural to attribute the difference to the epithelial island of Malassez which were regularly found towards the cementum. These epithelial islands showed only slight degeneration and they may act either as growth stimulators or play a protective role. Such an explanation is also supported by the observation that the basement membrane towards the epithelial cuff and the oral mucosa as a rule were the last collagen structures to be broken down.

The epithelial islands may also protect the cementum and the dentin against osteoclassic resorption. Resorption of the roots was not found on any of the permanent teeth, even when the alveolar wall was extensively broken down. It is known from replantation experiments, that cementum and dentin are resorbed just as easily as bone, thus the integrity of cementum must be maintained by some local protective factor. There is no basic difference between the alveolar and the cemental side of the periodontal membrane except for the presence of the epithelial islands adjacent to the cementum. Therefore, these islands must have a protective function.

The epithelium was not affected by the C-avitaminosis to the same extent as bone and connective tissue. In many instances the epithelial cuff was found to be quite normal in spite of the fact that during life the pocket could be blown open with the slightest jet of air. Therefore, an inferior quality of the epithelial cuff does not seem

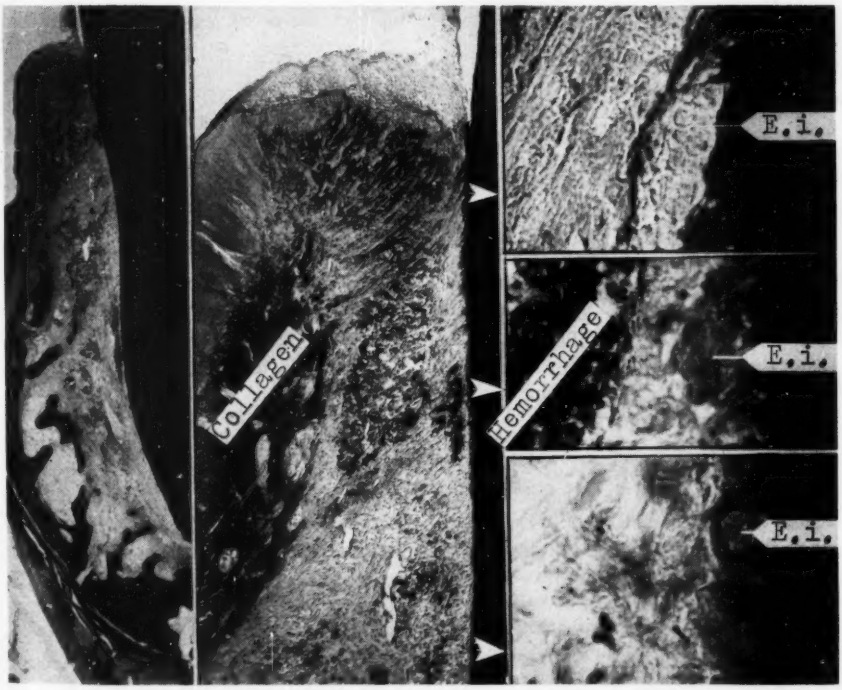


Fig. 5. Upper right permanent central incisor (Mallory). Cortical alveolar wall completely destroyed (left). Collagen nearly completely broken down except for area adjacent to oral mucosa (center). No downgrowth of epithelium below cemento-enamel junction. Collagen fibers extending from cementum best preserved below cemento-enamel junction (top right) and decreasing in length apically (center and bottom right). E.i. epithelial islands.

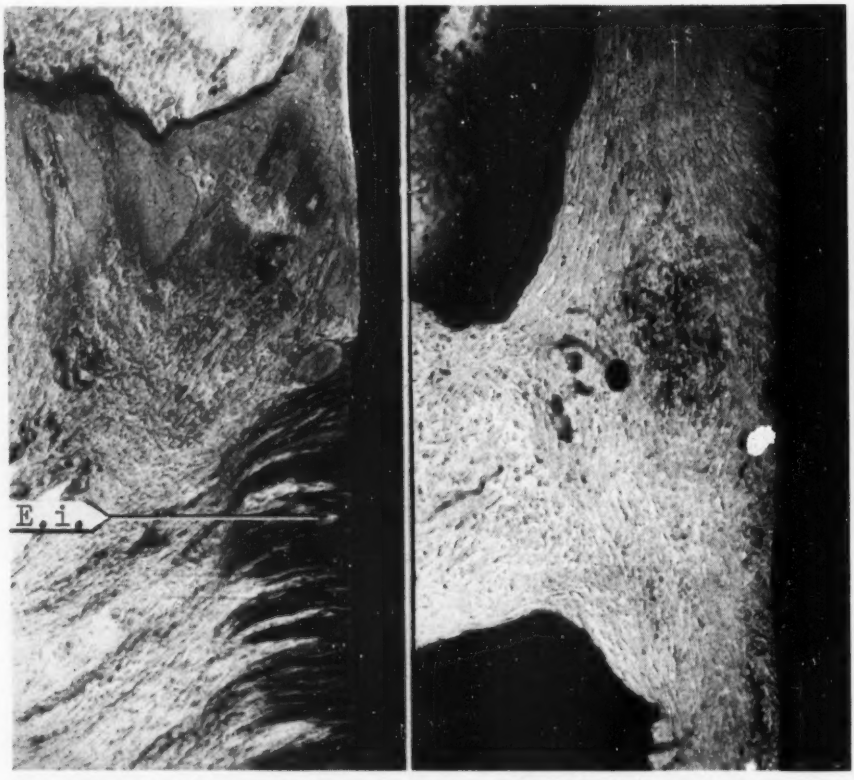


Fig. 6. Upper left primary cuspid (Mallory). Very short epithelial cuff with rather well preserved periodontal fibers just below (left). E.i. epithelial islands. Complete destruction of periodontal fibers adjacent to alveolar bone, but some fibers are seen adjacent to cementum (right).

to be a dominant factor in the pathology of scurvy. However, the clinical observations during toothbrushing clearly revealed increased bleeding, which shows that the epithelial cover was not as strong as normal. To what extent this was caused by the destruction of the underlying connective tissue cannot be decided. On the whole, the unfavorable conditions at the gingival margin in scurvy seem mainly to be attributable to the flabby consistency caused by the destruction of the collagen. This flabby consistency is also, most likely, responsible for the retraction of the gingival margin which was observed after death (Figs. 4 and 5).

The toothbrushing resulted in less gingivitis on the brushed side as compared with the unbrushed side (Figs. 8 and 9 are from brushed teeth). Thus, it was shown that the presence of bacterial plaque influences the pathologic picture. However, toothbrushing did not entirely prevent gingival inflammation. The reason for this may be that the rubbing necessary to remove the plaque was so strong that the gingival margins were injured mechanically.

The influence of occlusal stress represents an interesting incidental finding of this series. Increased mobility occurred as an early symptom of periodontal involvement, and became steadily worse with time. The intake of food cannot have been responsible for the loosening of the teeth as the

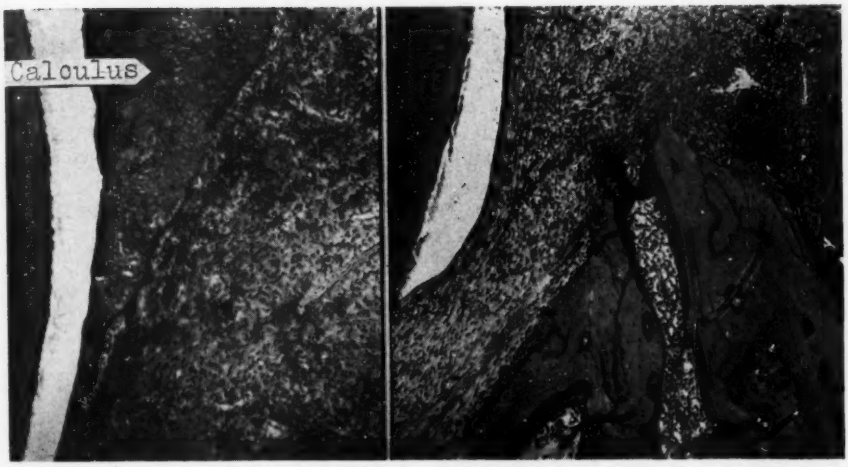


Fig. 7. Erupting upper left second premolar (hematoxylin eosin). Downgrowth of calculus (left). Epithelium adjacent to calculus destroyed. Severe infiltration of round cells in connective tissue. Marginal inflammation extending to alveolar crest (right).

diet (milk) required no chewing. This shows that even a reduced function can cause loosening of the teeth when the supporting structures are damaged by the vitamin C deficiency. The occlusal stress may partly have been responsible for the extensive resorptions of the alveolar wall in some cases (Figs. 5 and 6), but it apparently did not lead to a pocket deepening, since the periodontal fibers just below the epithelial cuff always appeared to be the strongest ones. Therefore, the increased mobility in scurvy cannot be compared with the increased mobility in the later stages of periodontitis in man, where extensive areas of periodontal fibers are destroyed by pocket deepening. More evidence has to be provided before it can be concluded that occlusal stress plays a role in the destruction of the periodontal structures in scurvy.

In the literature on experimental scurvy it has often been maintained that loss of teeth is an early symptom. In this series the experiment was started at an age when many of the primary teeth were present. During the observation period some of these teeth (but not all of them, Fig. 1) were lost. However, none of the permanent teeth was exfoliated. Experiments with monkeys have usually been started when the animals are young, and the loss of teeth reported in scurvy experiments has most likely been normal shedding of primary teeth, as it is rather difficult to distinguish between their primary and permanent teeth. Therefore, a more damaging effect may have been attributed to the vitamin C deficiency than is justified. The fact that so many primary teeth remained in place in this series suggests that instead of being hastened, the shedding of teeth is retarded in scurvy. This may be due to a retarded development of the permanent teeth. However, it is remarkable that the conditions of the periodontal membrane as a rule were better around the primary teeth than around the permanent ones. The explanation may be that the erupting permanent teeth constantly are changing their position.

It is known from previous experiments that healing in scurvy is dramatic and complete,[10] and there is reason to believe that all the damaged periodontal structures in this series would have been restored if vitamin C had been administered. Exceptions would have been the areas where epithelial downgrowth had occurred. Therefore, it is not likely that a shortstanding

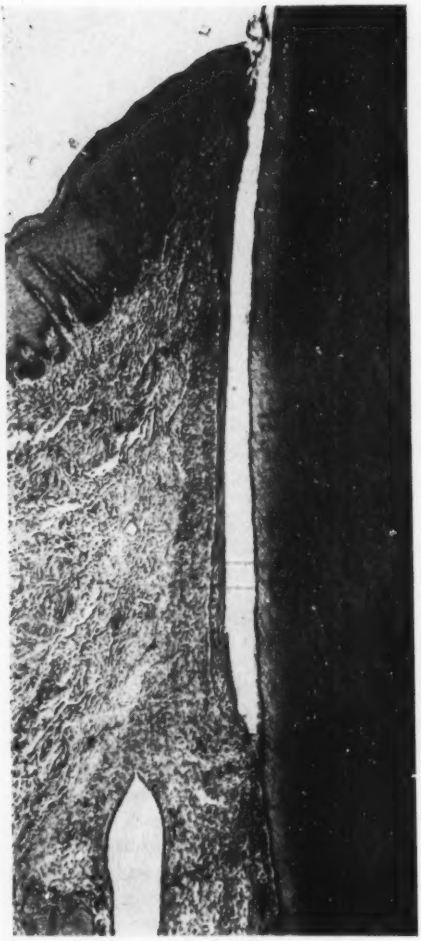


Fig. 8. Upper left permanent lateral incisor (hematoxylin eosin). Normal epithelial adherence to enamel. Only slight round cell infiltration in marginal third. No downgrowth of epithelium below cemento-enamel junction.

Fig. 9. Lower left permanent incisor (hematoxylin eosin). Normal epithelial adherence to enamel. Epithelial cuff approximately normal although some degeneration of cells are seen in deepest third. Slight inflammatory reaction in upper third.

vitamin C deficiency will produce very much permanent damage.

The clinical appearance of the periodontal structures in severe scurvy resembles in many respects that of periodontitis ("pyorrhea") in man, being characterized by bleeding gums and increased mobility of the teeth. However, the histologic picture is different. In severe scurvy the damage in the periodontal membrane is generalized, whereas in pyorrhea it is localized to areas below the deepened pockets. Increased mobility of the teeth in scurvy is facilitated by the generalized destruction of the collagen of the periodontal membrane and the resorption of the inside of the alveolar wall, whereas in pyorrhea it is facilitated by pocket deepening and localized destruction of the periodontal membrane.

Periodontosis (diffuse atrophy) is supposed to be a non-inflammatory destruction of the periodontal fibers below the "epithelial attachment," with ensuing downgrowth of this, initiated by an inferior

quality of the cementum. The histologic appearance of the periodontal structures in scurvy, however, does not correspond to this description either. It should be kept in mind that the periodontal fibers adjacent to the cementum in general, and those immediately below the "epithelial attachment" in particular, were the structures least affected by scurvy.

SUMMARY AND CONCLUSIONS

Four young monkeys were placed on vitamin C deficient diet for periods varying between 42 and 257 days. Clinical signs of scurvy appeared after about 90 days. The histologic examination after 257 days revealed nearly complete destruction of the collagenous fibers of the periodontal membrane, increased osteoclastic resorption and hemorrhages. Most of the primary teeth present at the beginning of the experiment were still there at the end of it.

From this series the following conclusions can be drawn:

1. The contention that loss of teeth is an early symptom of scurvy may depend on normal shedding of primary teeth having been taken as loss of permanent teeth.

2. Clinical signs of scurvy are bleeding from the gums and increased mobility of the teeth. Loss of permanent teeth seems to be a rather late symptom.

3. The histologic characteristics of scurvy are disturbance of dentin and bone formation, destruction of the collageneous fibers, increased osteoclastic resorption and capillary bleedings.

4. The Sharpey's fibers inserted into the cementum are not destroyed to the same extent as those inserted into the alveolar bone, depending, most likely, on some protective activity of the epithelial islands of Malassez.

5. The absence of osteoclastic resorption on the cementum must also be seen in relation to the presence of epithelial islands.

6. The periodontal fibers that are least affected by the C-avitaminosis are those just below the epithelial cuff and above the alveolar crest. This explains why downgrowth of epithelium did not occur more frequently.

7. The epithelial structures are not involved to the same degree as the connective tissue, although the epithelium is obviously weaker.

8. The first stage of increased mobility seems to be facilitated by the destruction of the periodontal fibers, while the extreme mobility in the later stages seems to be facilitated by the resorption of the alveolar wall.

9. The histopathologic picture of the periodontal structures in C-avitaminosis does not resemble that of periodontal disease in man.

REFERENCES

1. Boyle, P. F., Bessey, O. A. and Wolbach, S. B.: Experimental production of diffuse alveolar bone atrophy type of periodontal disease by diets deficient in ascorbic acid. J.A.D.A. 24:1768, 1937.

2. Crandon, J. H., Lund, C. C. and Dill, D. B.: Experimental human scurvy. New Eng. J. Med. 223:353, 1940.

3. Glickman, I.: Acute vitamin C deficiency and periodontal disease. J. D. Res. 27:9, 27:201, 1948.

4. Hart, C. and Lessing, O.: Der Skorbut der Kleinen Kindern. Stuttgart 1913.

5. Orban, B., Martin, W. B. and Hehn, R. M.: Histopathologic investigation of a case of scorbutic gingivitis. J. Periodont. 18:95, 1947.

6. Penney, J. R. and Balfour, Brigid, M.: The effect of vitamin C on mucopolysaccharide production in wound healing. J. Path. & Bact. 61:171, 1949.

7. Robertson, W. v. B. and Schwartz, B.: Ascorbic acid and the formation of collagen. J. Biol. Chem. 201:689, 1950.

8. Tomlinson, T. H.: Pathology of artificially induced scurvy in monkey—with and without chronic calcium deficiency. Publ. Health reports 57:987, 1942.

9. Turesky, S. S. and Glickman, I.: Histochemical evaluation of gingival healing in experimental animals on adequate and vitamin C deficient diets. J. D. Res. 33:273, 1954.

10. Westin, G.: Über Zahnveränderungen in Fällen von Skorbut bei Homo. Stockholm 1931, A. B. Fahlcrantz' Boktryckeri.

11. Westin, G. and Kalnins, V.: Experimental studies of the pathogenesis of marginal osteitis. Norske Tannl. Tid. 48:274, 1938.

12. Wolbach, S. B. and Howe, P. R.: Intercellular substance in experimental scorbutus. Arch. Path. & Lab. Med. 1:1, 1926.

Reattachment and Roentgenograms

by NATHAN FRIEDMAN, D.D.S.,* *Beverly Hills, California*

THE subject of reattachment in periodontal therapy has captured the imagination of clinicians and investigators since the turn of the century. Yet as a realistic result it is a matter of heated discussion, if not debate.[1, 6, 11, 15, 16, 18, 26, 27, 31, 41, 43] The preponderance of material offered as evidence of reattachment following periodontal treatment is based on clinical observation,[1, 8, 18, 22, 32, 34, 41] the next largest body of data is roentgenographic, [2, 5, 14, 36, 37, 38] and a relatively minute amount of proof is on a histologic basis. [4, 7, 33] The validity of each of these types of data is in reverse ratio to the order listed. The greatest amount of evidence offered (based on clinical probing) is the least valid, while the sparsest evidence (histologic) is the most convincing. Between these two extremes is the roentgenographic aspect of reattachment, which together with clinical measurements seems to be better balanced as far as amount of data and its merit.[3, 9, 10, 28] This aspect of reattachment evidence is to be explored in this paper. An evaluation of the clinical and histologic data must be made first. For many years proof of reattachment has been offered on clinical evidence that following periodontal treatment by subgingival curettage (non-excisive therapy), pockets were eliminated or reduced in depth. Sometimes these results were documented by pre-operative and postoperative measurements. This form of evidence has been contested widely and is obviously the weakest kind of substantiating data. Clinical probing is a highly subjective form of examination; the character of the tissue, the type of probe, the manual dexterity and manipulation of the probe by each operator, the reaction of the patient to probing, the postoperative change in the tissue, and the all too human quality of being more critical and exacting in preoperative evaluations than in postoperative assays makes this type of data subject to question. There is no way of knowing what the inability to probe a previously existing pocket really means. Reattachment, "closure" of a pocket, tight adaptation of the tissue—these are merely speculations to explain a clinical observation.

HISTOLOGIC EVIDENCE OF REATTACHMENT (ANIMALS)

Two types of reattachment are reported histologically: connective tissue and epithelial. Connective tissue reattachment is the more desirable because new bone, new cementum, and new periodontal ligament fibers are formed to create a new attachment apparatus where it had been lost due to periodontal disease (or experimental surgery). This type of reattachment occurs after successful treatment of the infrabony pocket. Sometimes connective tissue reattachment is referred to when new bone is not formed. This is especially true when a suprabony pocket is treated and the new tissues formed are cementum and periodontal ligament fibers. On the other hand, epithelial reattachment is the partial or complete eradication of a pocket by the formation of new epithelium that attaches to the root surface where no attachment existed before treatment. Generally this is conceded to be a temporary result, since the long epithelial attachment tends to detach coronally and a pocket reforms.

The most acceptable histologic proof of reattachment would be that in which the bottom of the pocket was clearly marked with a groove prior to or during the operation. Postoperatively new attachment coronal to the groove would be indisputable proof of success.

*Associate Professor of Periodontology and Oral Pathology, School of Dentistry, University of Southern California. This paper was presented at the 1957 Meeting of The American Academy of Periodontology, Miami Beach, Florida.

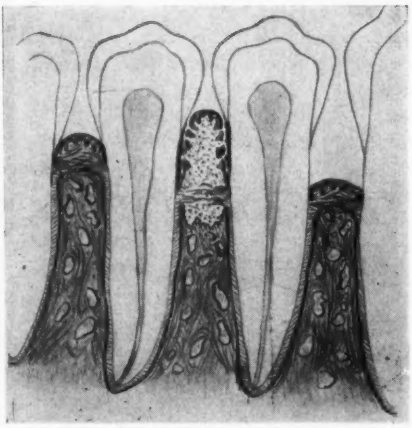


Fig. 1. Left side: Normal Periodontium. Center: Periodontitis—alveolar crest is lowered by resorption, the lamina dura over the crest is destroyed and the marrow spaces are enlarged and filled with inflammatory exudate. Right side: With the resolution of the inflammation the marrow spaces become smaller and a new lamina dura forms over the alveolar crest.

The histologic evidence of reattachment in human material is scanty. Experimental work on animals shows some success in reattachment attempts both in surgically created pockets without disease and pockets created surgically but permitted to become diseased. Ramfjord, treating epithelialized pockets in his experimental work on monkeys, reports reattachment in 16 out of 17 attempts (not complicated by lost bands, etc.).[29] The average amount of reattachment obtained was 2 - 3 mm. in the 16 cases. The average pocket depth pre-operatively was 8.8 mm., postoperatively it was 6.5 mm. The measurements made were clinical. No pocket measurements were made microscopically. The microscopic changes revealed that connective tissue reattachment occurred in some cases and epithelial reattachment in others. However, Ramfjord did not record what percentage of the cases were connective tissue in character and which were epithelial.

"In most cases it was possible from healed surface injuries on the root to identify microscopically the original depth of the surgically created pockets." Thus Ramfjord attempted to identify the bottom of the pocket. However, of the 17 pockets treated, in only 6 could the bottom of the pockets be identified microscopically with reasonable certainty. Several obscure factors appear in this report. In treating the pockets Ramfjord attempted to remove the crevicular epithelium, but makes no mention of the removal of the epithelium attachment. Suprabony and infrabony pockets were treated but the tabulated results do not indicate this. Despite the shortcomings of this work it is an important contribution to the histologic data on reattachment.

Linghorne and O'Connell reported soft tissue reattachment and alveolar regeneration in surgically detached tissues in dogs.[19] Reattachment following the surgical detachment of normal or healthy tissue has been reported many times in animals and humans.[19, 22, 35, 39] These are interesting results but not applicable to clinical situations where detachment is the result of disease and the operator is dealing with epithelialized pockets, non-vital cementum, and inflamed connective tissue. However, the third report by these investigators attempts to deal with this situation.[20] Epithelialized pockets were created in a manner different from Ramfjord's and these pockets were treated. The authors report that of the 15 pockets treated reattachment was gained in 14. Reattachment varied from 1 mm. to 7 mm., with the average being 4.8 mm. The pocket depth originally created was 10 mm.

These results would be impressive were it not for the inaccuracies in the experiment. (See appendix). After allowing for these inaccuracies, a re-evaluation of the results shows that in 11 out of 15 pockets treated reattachment of approximately 2 mm. was gained, in 2 pockets no change occurred, and in the remaining 2 the pockets deepened slightly. All of the measurements were made clinically and were not corroborated by microscopic measurements. This experiment adds little to the slim body of histologic evidence of reattachment.

HISTOLOGIC EVIDENCE (HUMAN)

Histologic evidence of reattachment in humans following treatment of pockets caused by periodontitis is meager and conflicting.

Schaffer and Zander reported microscopic evidence of reattachment and measured the amount both clinically and microscopically.[30] Eight pockets were operated but in only 6 were both clinical and microscopic measurements made. Of these 6, reattachment of 2 mm. was achieved in 3 cases. A fourth case showed reattachment of 1 mm. microscopically and clinical reattachment of 2 mm. This discrepancy in measurements is not explained. In the remaining 2 cases no reattachment was effected. The authors stated that both the connective tissue and epithelial type of reattachment was achieved, but they failed to tabulate how many of each resulted from their efforts. They also stated that both infrabony and suprabony types of pockets were treated, but did not indicate the type in their tabulation. As in the case of the animal experimentation, these authors did not indicate the bot-

tom of the pockets by grooving, thus having to estimate where they were originally.

In interpreting their sections they imply that the bottom of the pocket before treatment was at the point where the cemental curettage ended. The healing from this point to the bottom of the pocket after treatment was interpreted as new attachment. Actually, apical to the bottom of the pocket preoperatively was an epithelial attachment and transseptal fibers. These structures were completely or partially removed and the cementum to which they were attached probably was curetted. The curetted cementum shown in the section was apical to the original pocket, and the "new attachment" may have been in an area of surgical detachment.

Beube was fortunate enough to obtain a block section from a case treated fifteen years previously, and reported successful connective tissue reattachment.[6] However, no landmarks were present and no microscopic measurement was offered as to the probable amount of reattachment. That reattachment had occurred is indisputable, but the amount is open to speculation.

Ramfjord, in his review of the subject, has this to say about Box's case presented in 1928: "A review of Box's well-known work leaves one in doubt as to whether he demonstrated healing following surgical detachment by deep subgingival curettage, or whether the healing actually occurred on a previously denuded root surface. This doubt remains because the depth of the pocket was not measured from a fixed point on the tooth."[30]

Morris, who satisfied the criterion of marking the bottom of the pocket with a groove, reports results in conflict with those mentioned above.[24] Fifteen naturally occurring pockets were treated in an effort to gain reattachment. On the basis of clinical measurements 10 cases failed completely, in one case the pocket depth was increased 2 mm., and in the remaining 5 pockets the depth was reduced by 1 mm. in 3 and ½ mm. in the other 2. This reduction in pocket depth is contradicted by the microscopic findings. On histologic examination the author reports: "The positions of the nicks indicate that healing did not occur coronal to the bottom of the original pocket." He pointed out that only suprabony pockets on the labial surface were used. Clinicians usually agree that the infrabony pocket is the more favorable type in which to attempt reattachment,[15, 42] but Morris states this is open to conjecture.

From the foregoing brief review of a representative portion of the literature on the histologic evidence of reattachment, it seems reasonable to conclude that in spite of the shortcomings of the available data reattachment is possible. Morris' work which attempts to answer the criteria demanded for indisputable evidence contradicts the results of previous investigators, but this is not conclusive, since factors of pocket type, location, and mode of treatment may be significant. Both animal and human experimental data point to the fact that although some reattachment may be gained, it is not usual to eliminate the pockets by this mode of therapy. Very often in papers and discussions the mention of successful reattachment implies pocket eradication. If we accept a 2 mm. crevicular depth as within normal range, it would seem that the above implication should not be made (the reference to pocket eradication is in terms of depth and not as to whether or not the crevicular epithelium is ulcerated, a condition many investigators accept as the difference between a crevice and a pocket). The histological measurements of all investigators reveal that the average amount of reattachment effected was approximately 2 mm.

ROENTGENOGRAPHIC EVIDENCE

Some manner of assaying results must be utilized by the clinician and the clinical investigator. That clinical measurements alone have little validity is obvious. That roentgenographic changes alone following reattachment attempts are valid is challenged by many. It has been pointed out that in the roentgenogram the relationship between the soft tissue attachment and the alveolar crest is not determinable.[16] In addition, the author contends that the shape of the alveolar crest cannot be determined accurately by the roentgenogram. One cannot state unequivocally what portion of the bone has been resorbed, nor if what appears to be an infrabony pocket is in reality such a pocket. Even the use of radiopaque materials, such as silver or gutta-percha points, does not permit a diagnosis of an infrabony pocket to be made with certainty. The position of the point bucco-lingually in relation to the alveolar crest cannot be gauged. Interpreting the shape of a three dimensional structure by a two dimensional profile like the roentgenogram is fraught with inaccuracy.

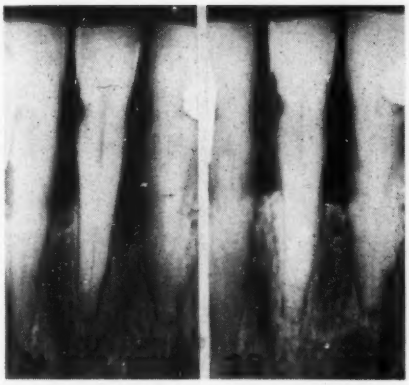


Fig. 2. 2A—Periodontitis with destruction of the lamina dura over the crest and enlargement of the marrow spaces. 2B—Postoperatively the resolution of the inflammation resulted in a new cortical layer over the crest and a decrease in the size of the underlying marrow spaces.

Very often the height of the alveolar crest is miscalculated because of the porosity due to the inflammatory changes. In short, the quantity may be confused with the "quality."

Another precaution which must be taken in diagnosing roentgenographic changes is one that for the most part has been overlooked. The apparent regeneration of the alveolar crest in a coronal direction in the roentgenogram does not always signify that this has occurred. Linghorne and O'Connell speculate that roentgenographic evidence of reattachment reported by many investigators where coronal growth of the alveolar crest occurred gradually was indeed such a coronal regeneration. They cite their experiment as the basis for their speculation, but a critical appraisal of this work leads to the conclusion that it is not valid, nor is the conclusion drawn from it valid. One must exercise caution and be keenly perceptive to interpret accurately from the roentgenogram what bone changes have occurred.

The best method available at this time to arrive at a presumptive conclusion that connective tissue reattachment has been achieved is by the correlation between clinical measurements and roentgenographic changes. A conclusion reached in this way may be valid if the clinician or clinical investigator is knowing and precise.

Carranza presented a carefully documented report in which a large number of cases were treated.[9] Pocket depths were recorded before and after therapy. He used root canal probes for greater accuracy in recording pockets. Clinical measurements showed varying degrees of "reattachment" in 40% of the cases. Roentgenographic changes listed as "marked repair of the osseous breach" occurred in approximately 15% of the cases. Some of the examples of roentgenographic changes are open to question, others show excellent results. The discrepancy between the clinical and roentgenographic changes is not discussed.

Cross attempted a careful clinico-roentgenographic evaluation.[10] Some of the results shown appear to be acceptable. In this report bone implants were used to gain reattachment in those pockets which he considered unsuitable for ordinary treatment methods. Residual pocket depth is accepted as a satisfactory result if clinical and roentgenographic aspects appear favorable. Analysis of the roentgenograms is rather unique, but here as elsewhere the apparent coronal regeneration of bone may be misleading.

Prichard has presented the most impressive roentgenographic evidence of bone changes where reattachment has been attempted.[28] Not only are more cases illustrated than in previous reports, but the results obtained are brilliant. In addition, these are not selected cases, but were reported in the chronological order in which they were treated. Prichard's report is a critical one. He is aware of the interpretative pitfalls of roentgenographic changes, the need for a careful technique in making roentgenograms, and correlating them with clinical probing. Although not all cases shown had pre- and postoperative measurements, the ones which were so documented give ample evidence of success.

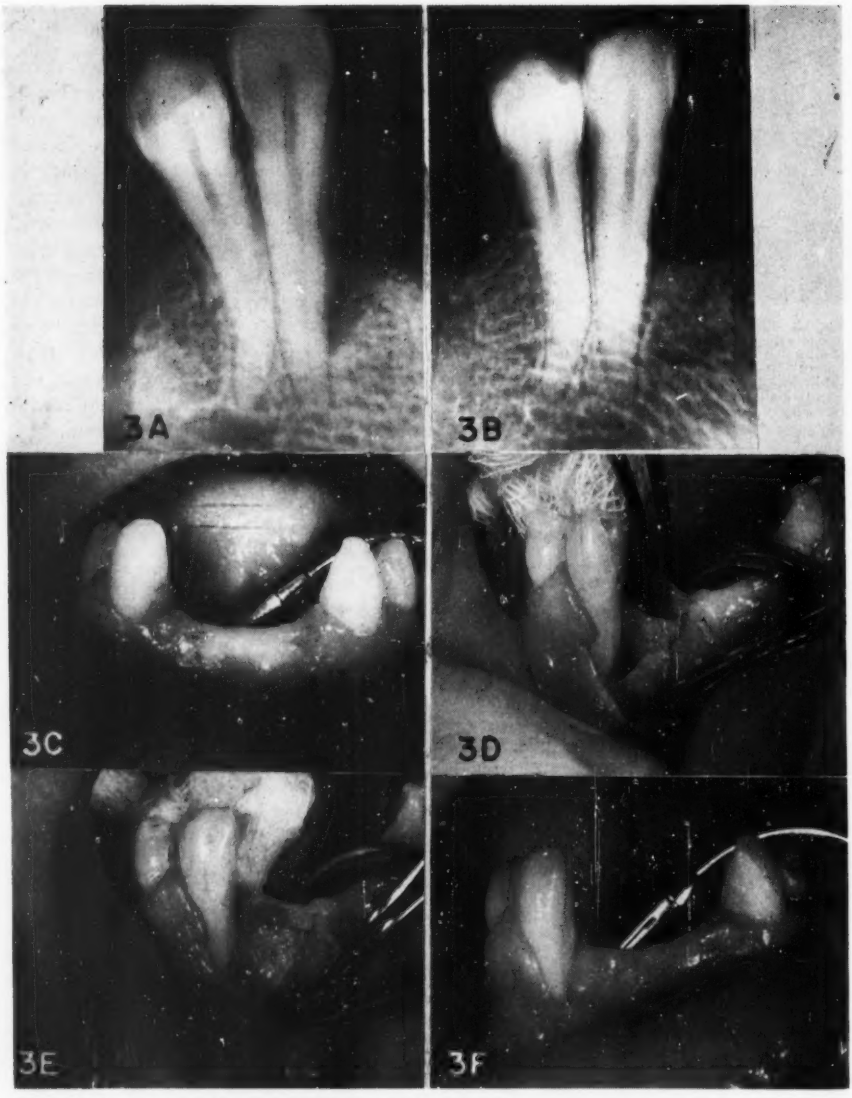
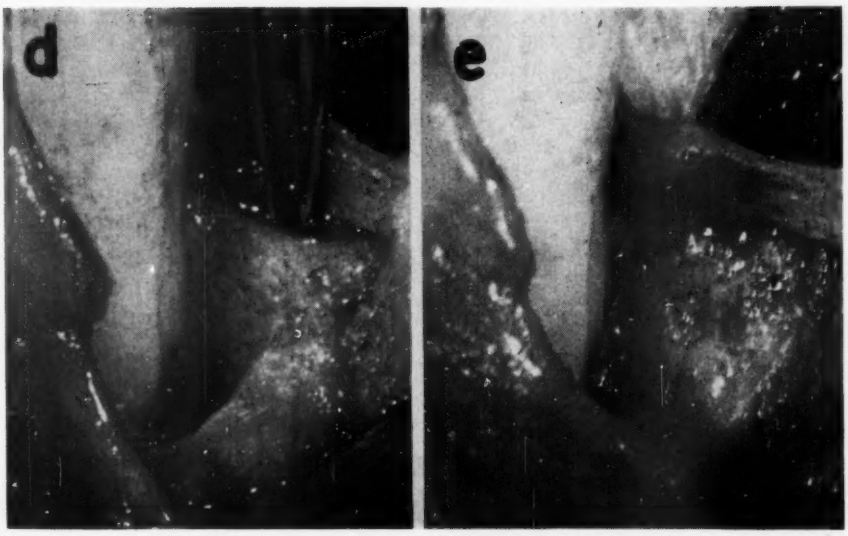


Fig. 3. 3A—Pre-operative roentgenogram. 3B—Postoperative roentgenogram—apparent coronal regeneration of bone is due to the decrease in size of the marrow spaces and a new cortical layer of bone. 3C—9 mm. pocket on the mesial of the cuspid. 3D—Flap back showing the "well" in bone. The inner wall of this "well" consists of enlarged marrow spaces. 3E—Osteoplasty performed—bone ramped. 3F—Postoperative result showing the gingiva taking the same form as the underlying bone.



3D and E—enlarged photographs of 3D and 3E.

ROENTGENOGRAPHIC CHANGES

Before speculating on what the roentgenographic changes in the following cases might signify, it would be well to consider the pattern of bone resorption that occurs in periodontitis. Weinmann, in discussing the progress of inflammation from the gingiva into the deeper structures of the periodontium, points out that the lamina dura of the alveolar crest interdentally is resorbed and the inflammation enters the bone marrow.[40] This would produce a radiolucent area on the roentgenogram at the crest. In addition to this, however, it is believed that osteoclasis occurs within the marrow spaces invaded by the inflammatory process, thus widening these spaces, [13,25] (Fig. 1). On the roentgenogram this would be revealed as an increased radiolucency and the alveolar crest would take on the cupped-out appearance of periodontitis. In examining a roentgenogram, therefore, we must bear in mind that the cortical layer (lamina dura) of bone over the alveolar crest is destroyed, and that the underlying marrow spaces have been exposed and *enlarged*, thus decreasing markedly the "density" of the alveolar crest area roentgenographically. When the periodontitis is eliminated by treatment the process of destruction is reversed. The marrow spaces become smaller and a new cortical layer of bone is laid down over the alveolar crest, thus increasing the "density" of the roentgenogram in this area (Fig. 2). This increased density, due to the changes in the marrow and the overlaying of a new cortical plate, is an occurrence that can lead to much misinterpretation of the roentgenograms. When these changes are confined to the interdental area at the tip of the alveolar crest, little difficulty is encountered in determining what has occurred. This is not true, however, when the bone resorption involves part of the alveolar crest and portions of the buccal or lingual plates as well.

Let us now consider Fig. 3. Roentgenographically Fig. 3A can be interpreted as an infrabony pocket, as in fact it was. Let us assume that the pocket was treated to gain reattachment and Fig. 3B was offered as evidence to support the contention that reattachment had been effected. It would

seem that the bone had regenerated in a coronal direction and a considerable amount of reattachment may have occurred. Has this happened? No. On the contrary, there is less bone postoperatively than there was pre-operatively (Figs. 3D and 3E). An osteoplasty had been performed and the rim of bone seen in Fig. 3D was removed.[12] The bone was ramped from the lingual to the labial and from the edentulous area toward the mesial surface of the cuspid root. That these slopes still exist may be supposed by the position of the overlying gingiva (Fig. 3F). What then accounts for the marked change in the postoperative roentgenogram? Note first the pre-operative roentgenogram and its clinical counterpart, Fig. 3D. As a result of the inflammation (perhaps complicated by other factors such as occlusal trauma) the bone had resorbed in such a way that an infrabony pocket existed on the mesial of the right cuspid. There was a "well" in the bone. The inner surface of this "well" had no cortical layer, and it is safe to assume that the marrow spaces were enlarged. In doing the osteoplasty the rim of the "well" was removed (Fig. 3E). With the elimination of the irritant and the pocket, the inflammation subsided and repair resulted. The enlarged marrow spaces that lined the "well" became smaller as a result of osteogenesis, and the entire area of the inner surface of the "well" and the contiguous area operated was overlaid with a cortical layer of bone. There was no coronal regeneration of bone, merely an increased thickness of the trabeculae bordering the narrow spaces, and a deposition over this of a dense cortical layer. This increased density appears on the postoperative film as a coronal regeneration of bone. Briefly, one would say that the quantity of the bone had not increased materially, but that the "quality" of it had changed greatly.

Consider the pre- and postoperative roentgenograms shown in Fig. 4. Notice that in Fig. 4A the lamina dura can be followed up the distal aspect of the bicuspid root to slightly above the bend. Postoperatively it would appear that the alveolar process had regenerated coronally. It might be claimed that some reattachment had occurred on the distal of this root. Actually, an osteoplasty was performed.[12] The pocket was eliminated by a combination of soft tissue and osseous surgery with no attempt to gain reattachment. The explanation for the roentgenographic changes in the bone is the same as that offered in the previous case. However, here the bone sloped from the buccal to the lingual. The entire slope was denuded of cortical bone and made radiolucent by the enlarged marrow spaces. With healing this large expanse was rendered more dense by the marrow changes and the new cortical layer covering the "roof" of the slope.

The peculiar funnelling effect of the alveolar crest over the distal of the bicuspid in Fig. 4B is frequently noted in roentgenograms offered as evidence of reattachment. It is probable that the apparent curve of the lamina dura to the distal is not a widened periodontal ligament space, but a confluence of cortical layers. Visualize the bone in Fig. 4A as if it were being viewed from the lingual. It would be seen as a slope running downward from the buccal to the lingual. The roof of this slope would consist of open marrow spaces giving a honeycombed appearance. Imagine an x-ray passing through this porous slope. The profile created would show a lamina dura on the distal of the root ending when it reached the porous slope, and the slope would show the radiolucency. Postoperative visualization would show that this porous slope had disappeared and in its stead is a slope of dense cortical bone. If an x-ray were directed through this it would throw a profile of lamina dura, making it appear as if it were going vertically up the distal of the root, when in reality it is the profile of the cortical bone of the slope. This dense slope blends with the slope coming from the edentulous area, and this confluence gives the effect of the funnel.

The roentgenograms shown in Fig. 5 show a marked change postoperatively. It would appear that there has been coronal

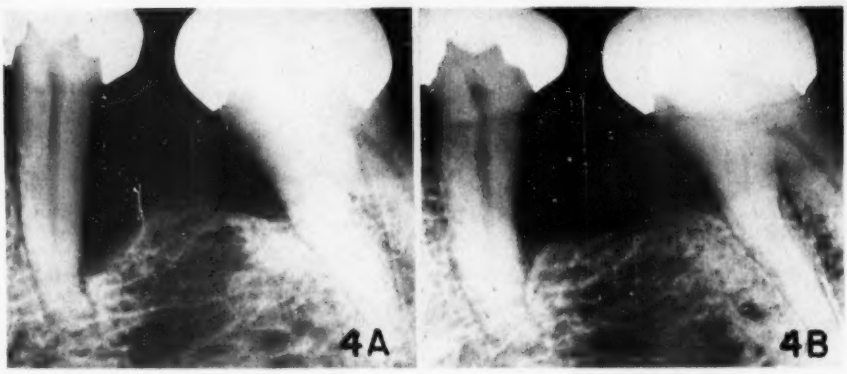


Fig. 4. 4A—Pre-operative roentgenogram—bone slopes from buccal to lingual on distal of the second bicuspid. 4B—Postoperative osteoplasty—narrowing of the marrow spaces and newly overlaid cortical layer of bone gives the appearance of regeneration of crest in a coronal direction.

regeneration of bone. One might assume that an infrabony pocket was present on the mesial of the cuspid and that the bone has regenerated coronally following a reattachment attempt. This roentgenographic change occurred following a gingivectomy. How can this change be explained? Note in Fig. 5A that the dim outline of the labial plate of bone can be seen considerably higher than the lingual plate. A 9 mm. pocket was present on the lingual aspect of the interdental area, going around to the lingual of the cuspid and to a lesser extent, the lingual of the lateral. This means that the bone was resorbed in such a way that a steep slope existed from the labial crest down to the lingual plate. The entire surface area of the slope was denuded of cortical bone and the marrow spaces were exposed and enlarged (Fig. 5C). With the resolution of the inflammation the marrow spaces became smaller, the entire slope surface was overlaid with a cortical layer, and the density of the bone was markedly increased (Fig. 5D). In the roentgenogram this change in density (quality) might be interpreted as coronal regeneration of the alveolar crest, but of course it is not.

It is interesting to note in Fig. 5B the two condensed areas mesial to the cuspid. One might speculate that these dense areas appeared because of "plugged" marrow spaces, or marrow spaces obliterated by bone. When roentgenograms of bone changes such as seen here are examined over a period of time, it is noted that the spongiosa architecture changes from time to time.

DISCUSSION

The purpose of this paper is to focus attention on the need for sharp, critical evaluation of the results obtained when reattachment is attempted. Eradication of the pocket by reattachment represents the pinnacle of success. It is the tendency of almost all clinicians to permit their critical faculties to soften when evaluating postoperative results. In re-examining some roentgenograms that previously were interpreted by the author as substantiating a successful attempt at reattachment a doubt now exists. One must consider what constitutes a practical objective when attempting reattachment. What is meant by successful reattachment? Does it mean pocket eradication or does it mean that some reattachment has occurred? Obviously, no general rule will be applicable. If one were to operate a pocket 10 mm. in depth, gain 2 mm. of reattachment, and have an 8 mm. pocket remaining, this would hardly constitute success in treatment. Yet one may operate a case, have a postoperative result of 4 or 5 mm. crevice and accept this as satisfactory, if clinically it appears free

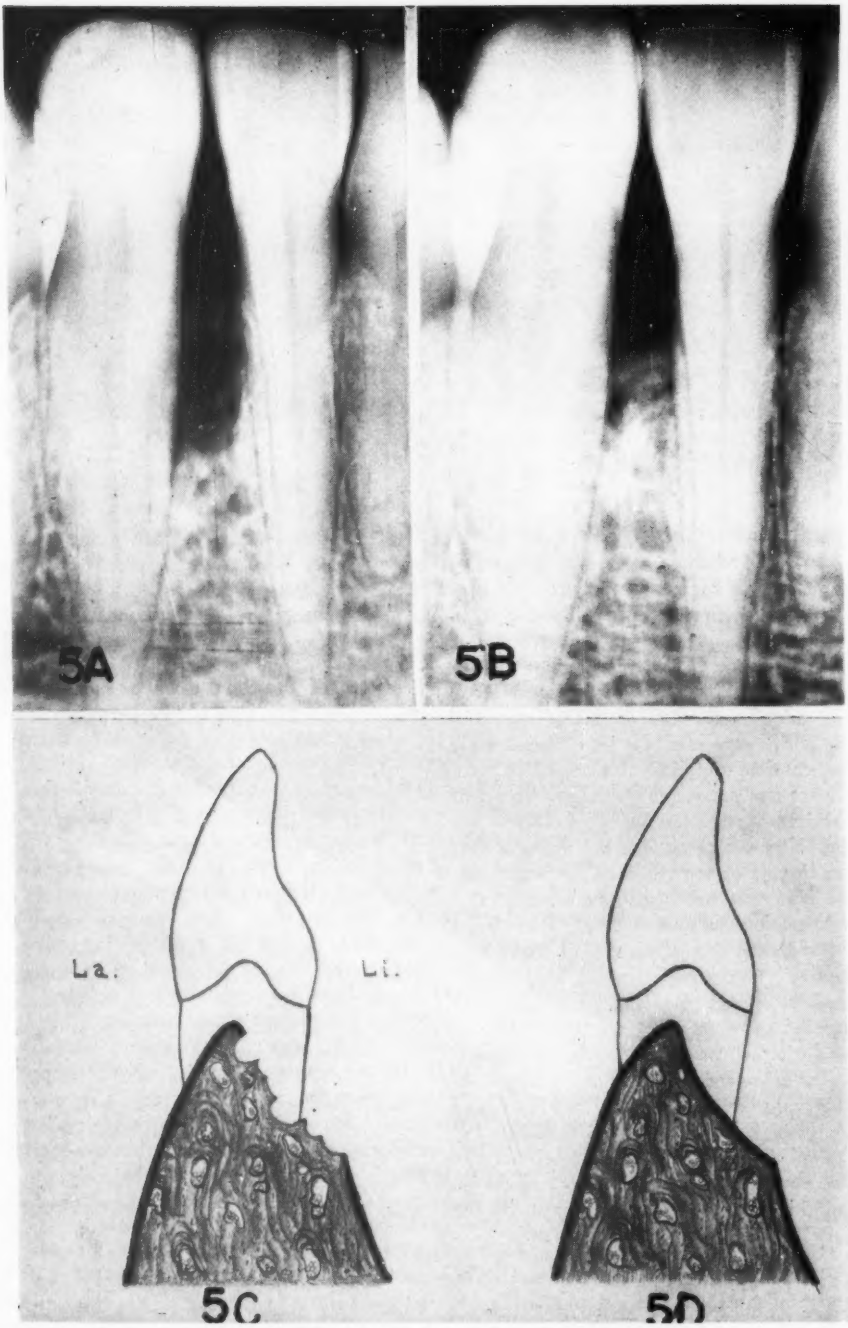


Fig. 5. 5A—Pre-operative roentgenogram—note dim outline of the labial plate of bone. 5B—Post-gingivectomy—bone appears to have regenerated in a coronal direction. This illusion is due to marrow changes and a new cortical layer deposited over the slope running from the labial down to the lingual. 5C—Drawing illustrates the author's conjecture of the pattern of bone resorption seen in Figure 5A. 5D—Changes in Figure 5B visualized in this drawing. Reduction in size of marrow spaces and overlaying of a cortical layer.

Fig. 6. 6A — Pre-operative roentgenogram—8 mm. pocket. 6B—Postoperative pocket depth was 4 mm. Reattachment was attempted and about 2 mm. was gained.

from disease and roentgenographically healing is apparent. Fig. 6 illustrates such a case. Reattachment was attempted here. Pre-operatively the pocket depth was 8 mm. and postoperatively 4 mm. This result was interpreted originally as reattachment solely on the basis of roentgenographic changes. However, in the light of information gained from evaluating the cases described above, caution is exercised in concluding that the roentgenographic changes were due solely to the coronal regeneration of bone. It is probable that some reattachment has been gained (about 2 mm.), but a portion of the change could be explained on the basis of marrow changes and the overlaid cortical layers.

Fig. 7 illustrated a similar but less definitive result. Here a lamina dura over the crest is not seen distinctly as in the previous case. Yet the roentgenographic changes are obvious. Again there appears to be coronal regeneration of the crest. Pre-operatively a 7 mm. pocket was present. The flap method was used, and postoperative measurement 2 years later revealed a depth of 5 mm. Little if any reattachment has occurred. The difference in the pocket depth is accounted for by the gingival marginal trimming and shrinkage. Note on the distal of the second bicuspid the destructive crestal changes give to the roentgenogram the appearance of a beginning infrabony

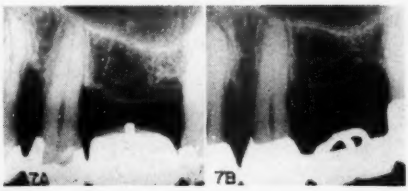


Fig. 7. 7A—Pre-operative pocket 7 mm. 7B—Postoperative pocket 5 mm. Reattachment attempt failed. Reduction of pocket was due to gingival marginal trimming and shrinkage.

pocket. Even here one could read into the postoperative result a coronal regeneration of bone, but healing produced a change in "quality" or density of the bone, and this accounts for the profile seen. In a previous paper the case illustrated by Fig. 9 was reported as successful reattachment therapy, meaning thereby that the pocket was eliminated.[12] Based on the clinical evaluation and the roentgenographic changes four years later, this still appears to be true.

CONCLUSIONS

The roentgenographic changes occurring after treatment which simulate those in reattachment must make us realize that conclusions reached by roentgenographic evaluation alone must be guarded. Until we learn in some way to distinguish between what is and what is not regeneration of bone in coronal direction, we must rely upon accurate clinical measurements correlated with the roentgenogram.

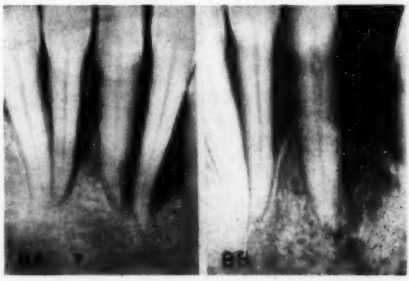


Fig. 8. 8A—Pre-operative pocket on mesial 1/ was 9 mm. 8B—Postoperative reattachment attempt resulted in 2 - 3 mm. of reattachment. Note the apparent coronal regeneration of bone where /2 was extracted.

Fig. 9. 9A—Crater in bone. Pocket 3/ 8 mm. 9B—Reattachment attempt successful.

The discrepancy between the clinical and microscopic measurements found in Schaffer and Zander's and Morris' work, and the discrepancy between the clinical and roentgenographic findings in Carranza's work indicate that probing is not completely reliable. The pocket pre-operatively is easily probed even with large instruments. The gingiva is usually inflamed and somewhat retractable. After treatment gingival and circular fibers regenerate, so that the gingiva is held firmly against the tooth. Even if no reattachment has been effected, entrance into the pocket is very much more difficult with the probe (or silver points). Even with the use of a thin probe, much care is required to get accurate readings. However, if such measurements seem to indicate new attachment and a critical assay of the roentgenogram parallels the clinical finding, we may assume that reattachment has occurred. Probing such areas repeatedly over a period of years may provide corroborative evidence that reattachment has been gained. It would be safe to assume that if mere adaptation resulted from the therapy (or epithelial reattachment), eventually this tight tissue would retract (or detach) and the probe would enter the pocket.

Both human and animal experimental data prove that reattachment is possible. However, as yet this data provides no statistically valid clue as to the frequency of its occurrence and the amount of reattachment. The histologic evidence offered shows the average amount of reattachment to be 2-3 mm. The clinico-roentgenographic evidence indicates greater success. This may be because the latter attempts were made with infrabony pockets. It is generally agreed that the infrabony pocket offers a much greater chance of success than does the suprabony pocket. Suprabony pockets were used in the histologic investigations in the majority of cases.

Prichard's results bring into focus an additional factor. He insists successful reattachment is predictable if one carefully

selects the *type* of infrabony pocket to treat. The high percentage of success he obtained gives credence to this demand of selectivity. The pocket should have three bony walls, the tooth forming the fourth wall. However this criterion makes the need for careful roentgenographic interpretation even greater. Can we readily distinguish roentgenographically a one wall infrabony pocket from a two and three wall pocket? Even if three bony walls exist, are they all at the same height? If not, then the postoperative result will have to be very carefully evaluated. The roentgenographic appearance of crestal regeneration may be partially due to density changes of a sloping bony architecture.

The determination of a three walled infrabony pocket requires careful clinical probing correlated with the roentgenogram. Sometimes the final determination must be left to visual inspection during the surgical treatment. Prichard does not treat the infrabony pocket by subgingival curettage. He removes all of the interproximal soft tissue and all of the soft tissue lying in the bony crypt. This approach is a surgical procedure. This departure in treatment methodology could well be significant as a means of achieving greater success in reattachment therapy.

SUMMARY

The clinical, roentgenographic and histologic evidence of reattachment is reviewed briefly and critically evaluated. Histologic data indicates that reattachment can be accomplished, the average amount being 2-3 mm. Roentgenographic evidence correlated with clinical measurements points to a greater success. This may be due to the fact that infrabony pockets were treated, whereas most of the reattachment reported histologically was achieved by treating suprabony pockets. Evidence based on clinical probing only has little validity.

The regeneration of bone in a coronal direction as evidenced by roentgenographic changes often is completely misleading. Cases of such apparent bone regeneration are shown which could be construed as successful reattachment. Not only was no attempt to gain reattachment made in these cases, but in some instances bone was removed. An explanation for these roentgenographic changes is offered. It is based on the fact that following the resolution of inflammation the marrow spaces become smaller (bone is laid down along the trabeculae) and a new cortical layer is formed. This increases the density of the bone in the roentgenogram. Under certain conditions these changes can be misinterpreted as coronal regeneration of bone.

Discussion centers about what constitutes successful reattachment and how it is to be interpreted. That reattachment can be attained is indisputable, but the amount is open to question. Recent evidence points the way to greater and more frequent success. It is also probable that success in the treatment of the infrabony pocket by reattachment may be predictable if the type of pocket is selected carefully.

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(The author wishes to acknowledge his appreciation to Carol E. Cooke, D.H., for the drawings).

REFERENCES

1. Barkann, L.: "In Our Opinion—Reattachment"—J. Periodont. Supplement to the Jan. 1943 issue, p. 7.

2. Bell, D. J.: "A Case of Reattachment?"—J. Periodont. 8:30 (Jan.) 1937.

3. Beube, F. E.: "Study on Reattachment of the Supporting Structures of the Teeth—J. Periodont. 18:55 (April) 1947.

4. Beube, F. E.: "A Radiographic and Histologic Study on Reattachment"—J. Periodont. 23:158 (July) 1952.

5. Bjorndahl, O.: "Reattachment and Bone Regeneration: Report of a Case"—J.A.D.A. 15:119 (Jan.) 1928.

6. Black, G. V.: Special Dental Pathology, 2nd Ed.—Medico-Dental Publishing Co., Chicago, 1920.

7. Box, H. K.: "Treatment of the Periodontal Pocket"—University of Toronto Press, Toronto, 1928.

8. Bunting, R. W.: "The Control and Treatment of Pyorrhea by Subgingival Surgery"—J.A.D.A. 15:119 (Jan.) 1928.

9. Carranza, F. A.: "A Technique for Reattachment"—J. Periodont. 25:272 (Oct.) 1954.

10. Cross, W. G.: "Bone Implants in Periodontal Disease"—J. Periodont. 28:184 (July) 1957.

11. Fish, E. W.: Paradontal Disease—J. B. Lippincott Co., Philadelphia, 1952.

12. Friedman, N.: "Periodontal Osseous Surgery: Osteoplasty and Osteoectomy"—J. Periodont. 26:257 (Oct.) 1955.

13. Goldman, H. M. and Cohen, D. W.: Periodontia—C. V. Mosby Co., St. Louis, 1957.

14. Goldman, H. M.: "Rationale for the Intrabony Pocket: One Method of Treatment, Subgingival Curettage"—J. Periodont. 20:83 (July) 1949.

15. Gottlieb, B.: "In Our Opinion—Reattachment"—J. Periodont. Supplement to the Jan. 1943 issue, p. 8.

16. Kronfeld, R.: Histopathology of the Teeth and Their Surrounding Structures—Lea and Febiger, Philadelphia, 1939.

17. Leonard, H. J.: "Periodontia Treatment for Maximum Reattachment" — J.A.D.A. 22:602 (April) 1935.

18. Leonard, H. J.: "In Our Opinion—Reattachment"—J. Periodont. Supplement to the Jan. 1943 issue, p. 5.

19. Linghorne, W. J. and O'Connell, D. C.: "Studies in the Reattachment and Regeneration of the Supporting Structures of the Teeth, I. Soft Tissue Reattachment"—J. Dent. Res. 29:419 (Aug.) 1950.

20. Idem: "III Regeneration in Epithelialized Pockets"—J. Dent. Res. 34:164 (April) 1955.

21. McCall, J. O.: "An Improved Method of Inducing Reattachment of the Gingival Tissues in Periodontoclasia"—D. Items of Interest 48:342 (May) 1926.

22. Merritt, A. H.: "The Pathology, Etiology and Treatment of Pyorrhea"—D. Cosmos 60:574 (July) 1918.

23. Morris, M. L.: "The Reattachment of Human Periodontal Tissues Following Surgical Detachment: A Clinical and Histological Study"—J. Periodont. 24:220 (Oct.) 1953.

24. Morris, M. L.: "Healing of Naturally Occurring Periodontal Pockets About Vital Human Teeth"—J. Periodont. 26:285 (Oct.) 1955.

25. Orban, B.: "To What Extent Should Tissues Be Excised in Gingivectomy"—J. Periodont. 12:83 (July) 1941.

26. Orban, B.: "In Our Opinion—Reattachment"—J. Periodont. Supplement to the Jan. 1943 issue, p. 8.

27. Orban, B.: "Pocket Elimination or Reattachment"—N. Y. State Dent. J. 14:227 (May) 1948.

28. Prichard, J.: "The Infrabony Technique as a Predictable Procedure"—J. Periodont. 28:202 (July) 1957.

29. Ramfjord, S.: "Experimental Periodontal Reattachment in Rhesus Monkeys"—J. Periodont. 22:67 (April) 1951.

30. Ramfjord, S.: "Reattachment in Periodontal Therapy"—J.A.D.A. 45:513 (Nov.) 1952.

31. Ritchey, B. and Orban, B.: "The Periodontal Pocket"—J. Periodont. 23:199 (Oct.) 1952.

32. Rule, R. W.: "Subgingival Curettage"—J. Periodont. 9:77 (July) 1938.

33. Schaffer, E. M. and Zander, H. A.: "Histologic Evidence of Reattachment of Periodontal Pockets"—Paradent. 7:101 (Oct.) 1953.

34. Shapiro, M.: "Reattachment in Periodontal Disease"—J. Periodont. 24:26 (Jan.) 1953.

35. Skillen, W. G. and Lundquist, G. R.: "An Experimental Study of Peridental Membrane Reattachment in Healthy and Pathologic Tissues"—J.A.D.A. 24:175 (Feb.) 1937.

36. Smith, T. S.: "Constructive Treatment of Diseased Paradental Tissues"—J.A.D.A. 22:1477 (Sept.) 1935.

37. Smith, T. S.: "The Treatment of Two Periodontal Cases"—J. Periodont. 20:129 (1949).

38. Stillman, P. R. and McCall, J. O.: A Textbook of Clinical Periodontia—Macmillan Co., New York, 1937.

39. Stones, H. H.: "The Reaction and Regeneration of Cementum in Various Pathological Conditions"—Proc. Roy. Soc. Med. 27:728, 1934.

40. Weinmann, J. P.: "Progress of Gingival Inflammation into the Supporting Structures of the Teeth"—J. Periodont. 12:71 (July) 1941.

41. Younger, W. J.: "Some of the Latest Phases in Implantation and Operations"—Dent. Cosmos 35:102, 1893.

42. Williams, C. H. M.: "Rationalization of Periodontal Pocket Therapy"—J. Periodont. 14:67 (July) 1943.

43. Williams, C. H. M.: "In Our Opinion—Reattachment"—J. Periodont. Supplement to Jan. 1943 issue, p. 10.

APPENDIX

Linghorne and O'Connell

Epithelialized pockets were created by laying flaps, removing bone and inserting periodontal packs between the flaps and the teeth before suturing. These packs were left in position for three weeks. Pocket measurements were made at the time of the detachment and when the packs were removed three weeks later. These epithelialized pockets were left undisturbed for three months, thus permitting them to become infected, thus simulating pockets resulting

from pathology. Measurements were made at the end of this time and the pockets were treated. The treatment consisted of packing the pockets with periodontal cement for 24 hours and then scaling the root surfaces. No attempt was made to remove the epithelial attachment or crevicular epithelium. Fifteen months later the animals were sacrificed. The result of the experiment revealed that of the 15 pockets operated, reattachment was effected in 14. Reattachment varied from 1 mm. to 7 mm., with the average being 4.8 mm. The pocket depth originally created was approximately 10 mm.

These results are invalidated by the fact that the measurements of the pocket depth 3 weeks after surgery (which is their starting point to gauge the amount of reattachment) are not acceptable as accurate. In attempting to create epithelialized pockets a pack was placed under the flap and left in position for three weeks. Yet in the report the investigators state: "Three weeks later the animals were again anesthetized and the pack, *if still present*,* was removed." Nowhere in the tabulation of the measurements is mention made whether the pack was or was not present. This means that where it was not present the operation may have been the equivalent of a surgical detachment without epithelialization, and the reattachment may have occurred by the time the measurements were made 3 weeks later. The investigators' own words further invalidate this experiment. They write: "The measurements from the groove in the tooth to the line of attachment shown in Table I 'at three weeks after surgery' *must not be taken as accurate.* The procedure was to insert the probe until a definite resistance was encountered, and then to record the measurement from the groove in the tooth. Short term studies to investigate early changes indicate that even if some reattachment has occurred, the new attachment would not yet have been strong enough to offer detectable resistance to the probe."

A re-evaluation of results would show that in 11 out of 15 pockets treated the measurements revealed that reattachment averaged approximately 2 mm., in 2 pockets no change occurred, and in the remaining 2 the pockets were deepened slightly. Since all of these measurements were clinical and made from the groove to the line of attachment ("After the 3rd month the line of attachment was found to be uneven and the measurement recorded was the distance from the groove to the most apical point.") —and no specific grooves or nicks were made to indicate the bottom of the pocket for microscopic measurements, the results at best can only be considered nebulous.

Nathan Friedman

*Author's italics.

Beg your pardon . . . . .

Our attention has been called to an error which occurred in an article by Dr. W. G. Cross on Bone Implants in Periodontal Diseases which was published in the July 1957 issue of the JOURNAL OF PERIODONTOLOGY (Vol. 28, page 184). In the review of one of the articles by Dr. H. Krømer the statement was made "In the second he stated that he had treated 13 cases of intrabony pockets with deep frozen stored bone" . . . the bone was not deep frozen but stored in 1:1000 merthiolate solution which was changed once a fortnight.

Therapy of the Incipient Bifurcation Involvement

by HENRY M. GOLDMAN, D.M.D., *Boston, Mass.**

WIDELY discussed in the periodontal literature is the problem of the therapy and prognosis of multirooted teeth involved by periodontal disease once the interradicular area has been affected to a degree that pocket formation enters into the region. A marked difference of opinion exists, one holding that such a clinical condition cannot be adequately corrected and that, therefore, the involved tooth should be extracted, whereas another is contrary, stating that these teeth should always be treated. The writer adheres to the latter view; however, it must be recognized that this statement is made to be contained within the bounds of feasibility and that the local environment, the degree of involvement, and the set of circumstances involved would constitute the formulation of a decision regarding the retention of such an affected tooth. Thus, careful preoperative examination and diagnosis are essential.

The purpose of this report is to describe a mode of therapy for the incipient interradicular involvement.

DIAGNOSIS AND PROGNOSIS

The prognosis of multirooted teeth with interradicular involvement is determined by the nature of the involvement (marginal and attachment apparatus), the topography of the pocket, the condition of the gingival tissue, the tooth relationships (with its neighbors and antagonists—roots of adjacent teeth close together), the anatomy of the tooth (roots fused or widely separated), the mobility pattern, and the set of circumstances at hand.

Often the interradicular area of a multirooted tooth may be affected by periodontal disease without any visible radiographic evidence. Should only a small entrance be present into the bifurcation area of a mandibular molar, then no change in registration in the radiograph would occur; usually it takes appreciable loss of alveolar process to elicit any deviation. Thus, clinical examination by probing is essential for a diagnosis. The latter, together with radiographic findings, offers a complete evaluation of the condition.

Of extreme importance in the prognosis of an interradicularly affected tooth is the nature of this involvement. The following questions may be asked: Has the disease process resulted in complete destruction of the interradicular tissue or has only one portion been affected? In the mandibular molar area, has the tissue between the roots been completely destroyed through and through, or is only one side involved? If so, to what extent? It is of importance to know not only how far toward the median line of the tooth the pocket has extended but also how deep and wide it is. Thus, three dimensions are important. In the maxillary molar teeth, three roots must be taken into consideration: the two buccals and the palatal. Usually a pathway exists between the two buccal roots extending towards either the mesial or the distal of the palatal root. Any topographical variation may exist; the two buccal roots may be involved or the mesial or the distal of the palatal may extend towards the two buccal roots. Another type of involvement may exist only on the proximal aspect of the tooth, extending to the opposite side. This is seen in the maxillary first premolar region.

The incipient bifurcation involvement may be described as an interradicular periodontal lesion in which pocket formation depth is minimum. When probing this lesion, the instrument is placed into the pocket and the topography noted. The interradicular entrance is probed to ascertain the depth. The anatomical features of the

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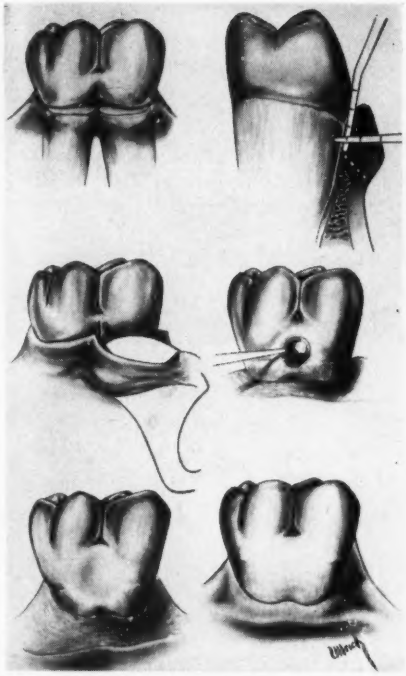

Fig. 1. Diagram of the step-by-step procedure to eliminate the incipient bifurcation involvement.

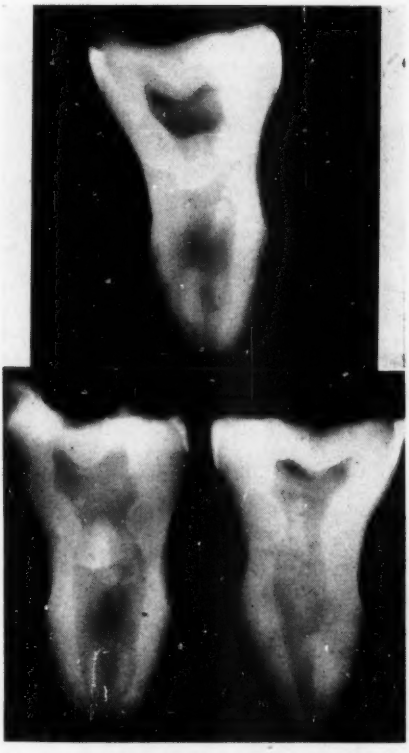

Fig. 2. Radiographs of extracted tooth which had been ground to ascertain the limitations of the procedure. Note that although the tooth had been cut to the outermost limits, there is still considerable distance to the root canals.

roots are determined to find out whether the roots are fused, close together, or whether they are widely separated.

THERAPY

Once the topography of an interradicular involvement has been ascertained, its management is planned. Generally speaking, therapy is identical to that which is applied elsewhere. The objectives would be a healthy gingival attachment, contoured to physiologic standards and stability of the affected tooth. The area should be self-cleansing as much as possible; also, the area must be capable of being cleansed by the patient so that no foodstuff or debris will accumulate. Not to be overlooked as well is a fastidious oral physiotherapy by the patient, for without this aid in cleansing, regardless of topography, failure may (will) result.

The proposed operative procedure is designed to obliterate the bifurcation involvement by removing tooth substance and soft tissue. In this way the diseased gingival attachment (pocket formation) and topography are treated to obtain a healthy attachment after healing; the area is then accessible and easily cleansed.

The lesion involving the interradicular area for which the proposed therapeutic plan is offered, is situated on the buccal or lingual surfaces of the mandibular molar teeth, and the buccal aspect of maxillary molar teeth. On the palatal aspect of these latter teeth, the involvement is on the proximal surface but usually close to the

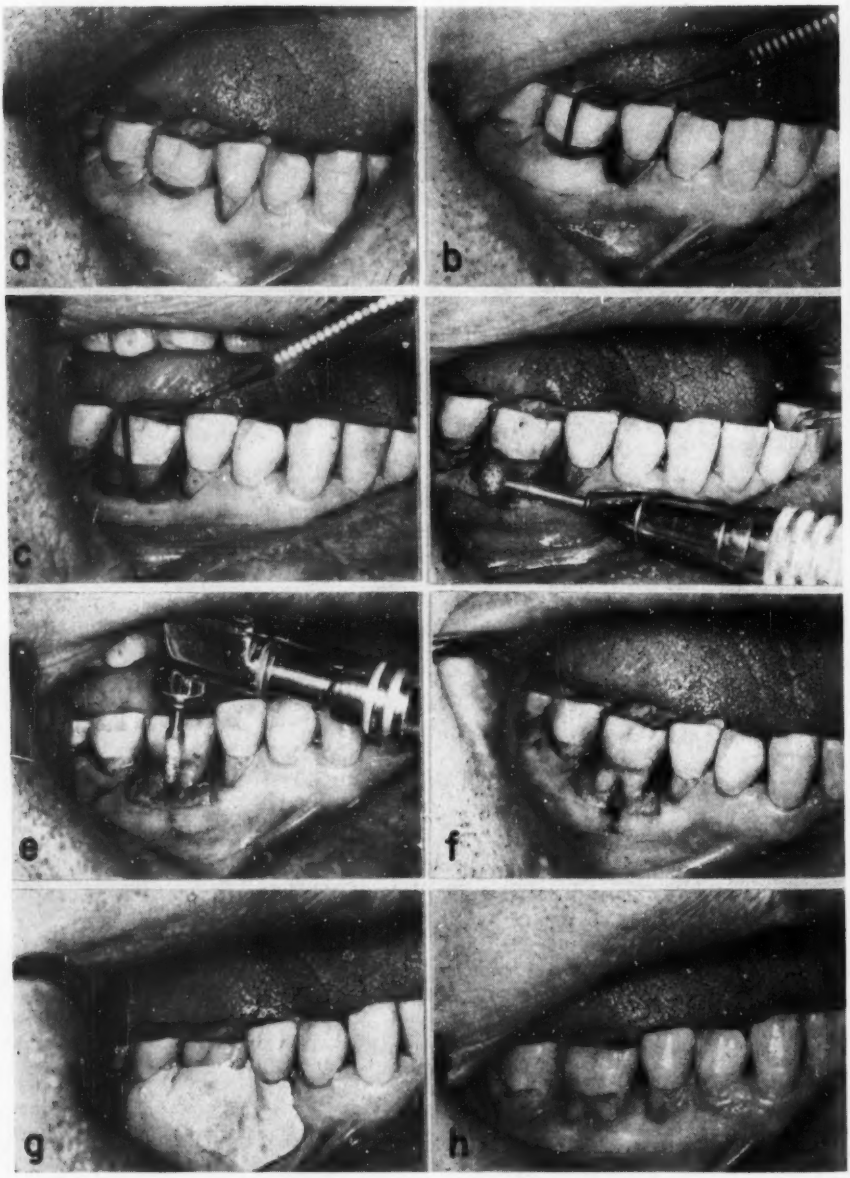

Fig. 3. Step-by-step procedure of the therapy of the incipient bifurcation involvement. Note the postoperative healing.

palate, depending upon the anatomy of the tooth and its position over the ridge (rotation, contacts of the teeth, etc.). If accessible, the proposed operation can be performed. Otherwise, it cannot. The lesion must be measured in three dimensions: the apical, the interradicular penetration, and the width. In conditions in which the pocket depth measures three to four millimeters, or even greater, and penetration is one millimeter or slightly over at the outset, the following therapy may be instituted:

After initial therapy is performed, under local anesthesia, a gingivectomy is done removing the soft tissue wall of the pocket. This excision of tissue conforms to the advocated procedure—bevelling, etc. Once the tissue is removed, the tooth roots are contoured to take out the anatomical V-shape, creating a saucer-shaped area. The crown is contoured as much as possible. The gingival margin is then festooned to desired form, making the margin flush with the tooth in the interradicular space. Thus the gingival tissue conforms closely to the tooth surface, indenting in the bifurcation area. Osteoplasty may be performed if necessary. The entire area is so shaped, soft tissue as well as tooth, to the desired form so that the area will be self-cleansing and easily reached by the excursion of the toothbrush in the oral physiotherapy procedure. The area is then covered by a periodontal pack.

The above operation is shown in Fig 1, a drawing of the step-by-step procedure. This illustration shows a bifurcation involvement on the buccal aspect of a mandibular molar. The examination discloses a pocket depth of three millimeters and an interradicular penetration of one millimeter. After the soft tissue wall has been excised, the tooth is contoured, the indentation made by the roots being entirely removed. Note that the ridge usually found at the cervical enamel junction has also been smoothened. The gingival tissue is festooned to indent into the interradicular area, resulting in a smooth accessible area. The entire crown of the tooth may have to be reshaped to give a desired occlusal-cervical convexity. This contouring of the tooth is termed "odontoplasty." The tooth surface is burnished with sodium silicofluoride paste which not only overcomes any sensitivity of the tooth but also gives the surface a glossy appearance. Should the tooth show any sign of sensitivity prior to the proposed operative procedure, then it should be desensitized first.

The deeper the penetration, the more cutting of the tooth and contouring of the gingiva are necessary. Thus, there is a distinct limitation to this procedure in that too much of the root surface may have to be cut. Up to one millimeter and perhaps slightly more penetration can be overcome by this procedure. Over this, it is not possible to eradicate the anatomical features which constitute the bifurcation involvement.

In order to ascertain an approximate extent to which a tooth can be ground, extracted molar teeth were examined. Measurements were made in the bifurcation area and the various anatomical features noted. Roots which were widely separated presented a different problem than those teeth which had roots fused together. The measurements revealed that most notches in the bifurcation area could be eliminated by grinding approximately one millimeter when the roots were fused. Teeth were ground and then radiographed to determine the extent of the grinding in relation to the root canal. Teeth with widely separated roots were ground approximately one millimeter but in this instance since there is no notch, each root had to be ground separately. (In the clinical procedure, gingival tissue fills this interradicular area and the tooth is ground to a point where the entire bifurcation area is filled completely with tissue.) Fig. 2 illustrates a tooth which was ground one millimeter and then radiographed. Note that there is still a great deal of distance between the surface and the root canal in both the mesial and distal roots.

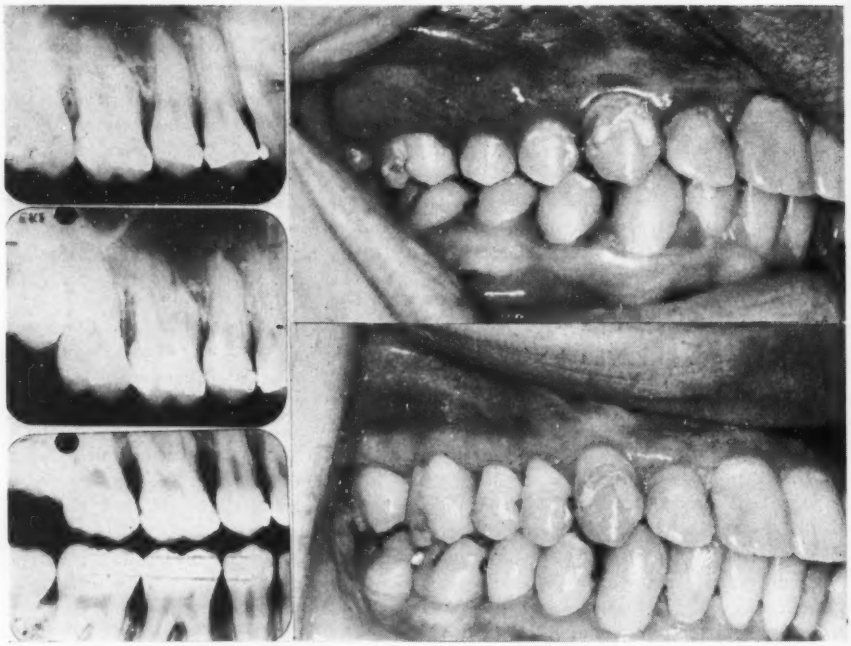

Fig. 4. Before-and-after photographs of the therapy of an incipient bifurcation involvement of the maxillary right first molar. Note the topography of the gingiva and its relationship to the crown postoperatively.

At times it is feasible to operate in this fashion, allowing for a certain depth of tooth structure in the bifurcation area to remain. It is an aid in this instance but does not eradicate the complicating topography completely.

Fig. 3 illustrates a practical case in which the bifurcation on the buccal aspect of a mandibular right first molar could be penetrated about one millimeter. Pocket depth measured four millimeters. The gingival margin had already receded so as to expose the cementum surface of the root. After gingivectomy (C) the probe could be placed interradicularly. With a diamond stone the gingival tissue and root surface were contoured making the gingival tissue flush with the tooth. In this instance it was necessary to bevel the margin of the bone process. Postoperatively the gingival tissue of the margin filled the interradicular area solidly and the pocket was thus entirely eliminated. The crown was then ground (F), the results seen in "G." A pack was then placed in position. Sixteen days later the gingival tissue had healed and the bifurcation area could not be penetrated by probe. The area could easily be cleansed as any other tooth with a receded margin.

Fig. 4 are before-and-after photographs of a maxillary molar area treated as described above. Note the contour of the first molar tooth and the anatomy of the gingival tissue in the after photograph. Radiographically no bifurcation involvement could be discerned.

SUMMARY

A procedure is herewith presented for the treatment of the incipient interradicular involvement. Its attributes consist of creating not only a self-cleansing tooth-gingival area, but also makes for accessibility in the oral physiotherapy procedure.

Experimental Occlusal Trauma Imitating Cuspal Interferences*

by FRANK M. WENTZ, D.D.S. PH.D.; JOSEPH JARABAK, D.D.S., PH.D. AND BALINT ORBAN, M.D., D.D.S., *Chicago, Illinois*

TISSUE changes produced by experimental occlusal trauma in animals have been studied extensively in the past.[1,2] The tissue changes in experimental animals correspond exactly to those found in human autopsy material.[3,4] However, objections and questions have been raised as to the validity of drawing conclusions from animal experimental material and relating these to tissue changes that occur in the human dentition. The primary objection was that in all previous experiments the trauma was applied in one direction only, with the result that the experimental tooth moved out of occlusion and then finally out of range of the traumatic forces. This form of response of a tooth to an applied force is analogous to orthodontic tooth movement. On the other hand, it has been claimed that the actual traumatic forces in the human are different than the "orthodontic like forces" produced previously in experimental animals. The claim is that in the human, unlike experimental animals, the traumatic forces act alternatingly in one and then the other direction. These forces have been termed "jiggling forces." A tooth subjected to these "jiggling forces" will tend to be moved by its antagonist to the buccal then to the lingual and then back again and again during the masticatory or lateral excursive movements of the mandible.

On the basis of this claim that the previous studies did not produce the "jiggling" type trauma observed in humans, an experiment to imitate this form of trauma was devised as follows:

MATERIAL AND METHOD

Six Rhesus macacus monkeys about four years of age, with permanent dentitions were employed in the study. The upper right second premolars were prepared for full crowns. The gold crowns were prepared in such a manner that the buccal cusp and its inclined plane was exaggerated. The crown was cast and temporarily fitted to position and adjusted so that in the median occlusal position and right lateral movements of the mandible the tooth was subjected to excessive occlusal stress in a *buccal direction*. The crown was removed from the tooth and a small hook was soldered to the palatal side. An orthodontic palatal arch wire was devised and fitted, utilizing the upper right and left second molars for anchorage. A small finger spring was soldered to the arch wire and adjusted to engage the hook previously soldered to the palatal surface of the second premolar crown. The crown and the palatal appliance were cemented to place. The finger spring was activated to exert (2 oz.) tension when engaged into the palatal hook of the crown. The tension of the finger spring upon the crown tended to pull the tooth to the lingual side each time the animal opened his jaw and disoccluded his teeth (Fig. 1).

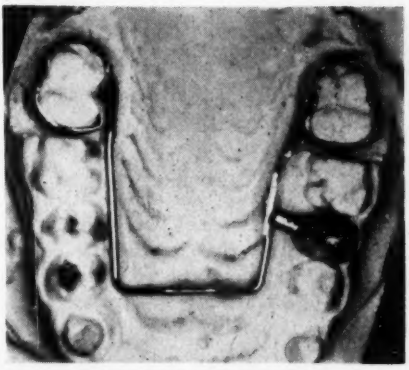

Fig. 1. Orthodontic appliance used in the "jiggling" experiment. Crown on 2nd premolar with lingual lock. Lingual arch with finger spring.

Two of the monkeys were sacrificed three days after the appliances had been activated. Of the remaining, one was killed at fourteen days, one twenty-one days, one at three months and one at six months.

*From Loyola University School of Dentistry, Chicago, Illinois. Presented at the 43rd annual meeting of Academy of Periodontology, Miami, Florida —October 31-November 2, 1957. Financial support for this investigation was obtained from the Southwestern Texas Dental Foundation (C. W. Finley).

All specimens were fixed in ten per cent formalin; decalcified and the upper right premolar areas sectioned serially in a bucco-lingual direction and the corresponding region of the lower jaw was sectioned also in the same direction. The left side, which was used as the control, was sectioned similarly. The sections were stained with hematoxylin and eosin, some with the mallory method; others by silver impregnation.

At the beginning of the experiment and at intervals, photographs were taken of the experimental and control sides of the jaws. This was done to record any changes that may have occurred.

THREE DAY MONKEYS

Findings: Three days after cementing the crown and palatal appliance, the tooth with the crown was slightly mobile and could be moved in a bucco-lingual direction. Histologic sections of the experimental tooth showed the following changes:

Buccal Side: Fig. 2 shows the tooth which was subjected to trauma for three days. There is a compression of the periodontal space at the buccal alveolar margin (Fig. 3) and at the apex of the buccal root (Fig. 4).

Fig. 2. (TM5-1) Experimental tooth of 3 day animal. a — labial alveolar margin, b — lingual alveolar margin, c — apex of the mesio buccal root, d — apical area of the lingual root.
Pressure necrosis at a and c. Resorption of alveolar bone at d. No changes at b. Width of periodontal space average .19 mm.

In both of these areas undermining resorption is occurring in the traumatized necrotic bone tissue. This resorption is well illustrated just lingual of the apex of the buccal root.

This experiment differs from previous high crown experiments in that the tooth does not exhibit a tipping. In spite of the necrotic areas, there is a certain width to the periodontal space. This finding is in accordance with the experimental design to "jiggle" the tooth in a buccal and then a palatal direction and not drive the tooth in one direction only.

It is remarkable that in a 3 day experimental period, resorption and repair processes have progressed to such an extent. Resorption is occurring at the alveolar crest margin (Fig. 3). At the apex of the buccal root not only necrosis and extensive bone resorption is occurring but new bone formation is in evidence on the endosteal surface of the alveolar bone. This new bone is of the osteophytic type. The bone marrow bordering these areas is fibrous in character. The characteristic cellular sequence of an inflammatory process is not apparent.

Similar findings were observed in the second, third day animal. In this short period of time, formation of immature bone tissue has also begun on the endosteal surfaces of the alveolar bone.

Palatal Side: The palatal side of this same tooth does not show excessive tissue changes of any kind. However, the alveolar crest in the second three day experimental animal, the entire periodontal ligament is necrotic due to the crush between tooth and bone. However, there is an intact area of connective tissue between the necrotic area and the end of the epithelial attachment (Fig. 5), without any inflammatory reaction. The periodontal space is of normal width in spite of the pressure necrosis caused by the jiggling of the tooth.

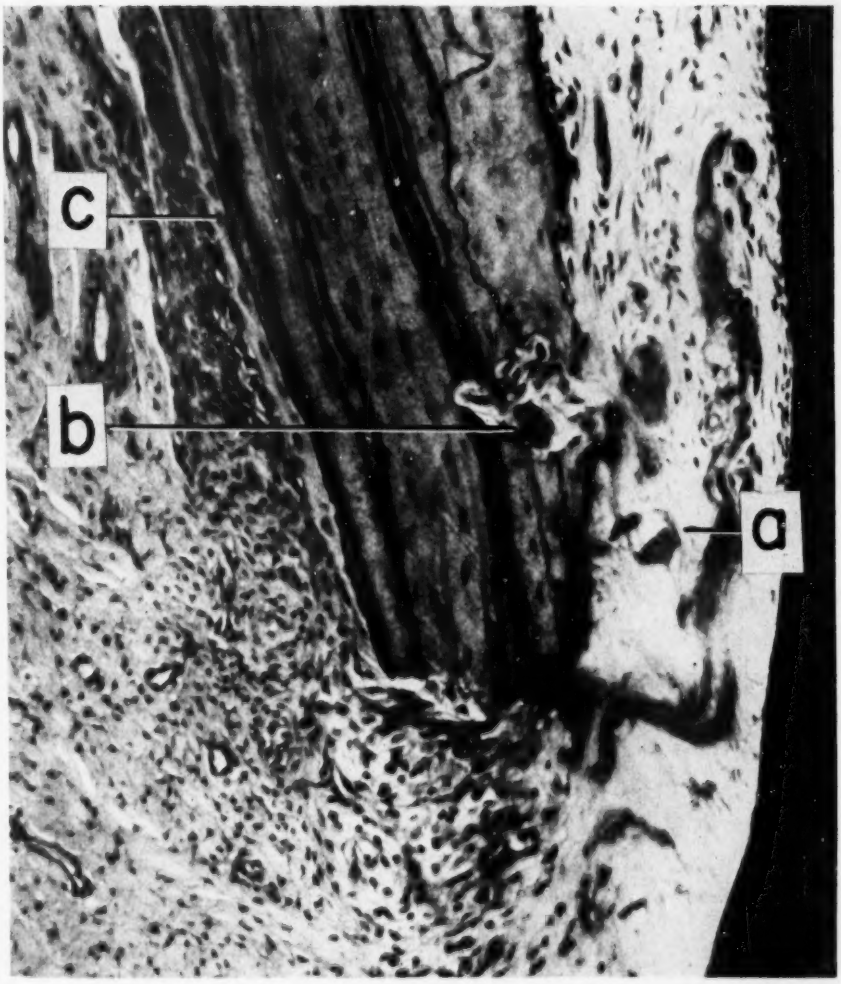

Fig. 3. (TM5-1) Higher magnification of pressure area (a) in Fig. 2. (a) necrotic periodontal ligament, with thrombosed blood vessels. Resorption of bone (b) apical to the pressure area. Some resorption is taking place at the tip of the alveolar crest. New layer of bone at the periosteal surface of the alveolar crest (c).

FOURTEEN DAY ANIMAL

Fourteen days after the crown and the palatal appliance had been inserted, the second right premolar was very loose. Clinically during the experimental period and at sacrifice, no imflammatory changes were observed in the gingiva of the experimental tooth.

The histologic sections showed the following:

Bifurcation: The fourteen day animal exhibited extensive destruction of the periodontal ligament, especially in the bifurcation (Fig. 6). Here extensive necrosis was observed on both the palatal and buccal surfaces of the inter-radicular septum. Un-

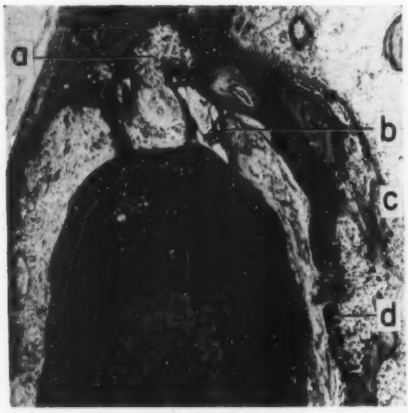

Fig. 4. (TM5-1) Apical area of buccal root (c of Fig. 2) with areas of compression (b) with undermining resorption of bone in different areas (a, d) and new formation of immature type of bone on the endosteal surface of the alveolar bone (c).

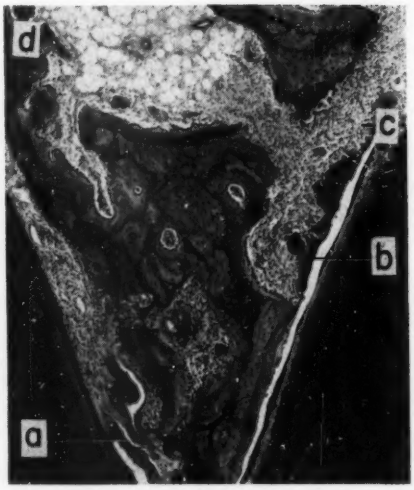

Fig. 6. (TM1-1) Fourteen day experiment. Area of bifurcation of the experimental tooth. Necrosis of the periodontal ligament on the buccal (a), as well as lingual (b) surfaces of the interradicular bony septum. c — resorption of root surface apical of compression area. d — immature (osteophytic) type of bone formation on the endosteal surfaces of bone trabeculae. The bone marrow near the compression areas is fibrous, but there is no inflammation present.

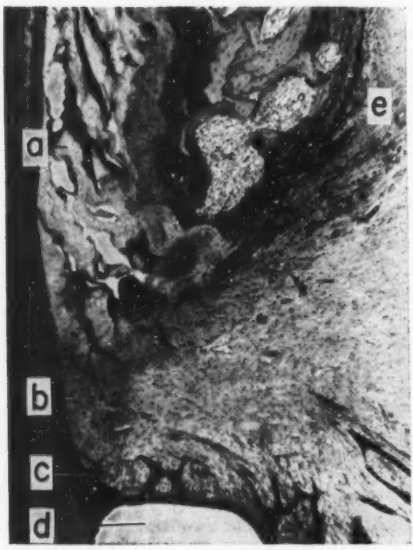

Fig. 5. (TM6-1) Three day experiment. Lingual alveolar crest with compression necrosis (a) of the periodontal ligament. Between necrotic area and epithelial attachment (c) uninjured fibers (b) of the dento-gingival junction. d — enamel, e — new bone formation on the periosteal surface of the bone.

dermining resorption of bone tissue, endosteal new bone formation, as well as resorption of the tooth is progressing simultaneously.

Apex: Due to undermining resorption most of the alveolar bone proper has been lost at the apical area of the socket. Instead of a lamina dura in this area the alveolar bone consists of small islands of bone tissue only (Fig. 7). These bone islands may show an osteoid seam at one border with Howship's lacunae and osteoclasts on the other. The interplay between bone apposition and resorption in this area is the result of the "jiggling" trauma. Resorption of the root of the tooth has also occurred.

None of these tissue changes are accompanied by the inflammatory cellular response which is part of a stereotyped inflammatory reaction. The picture is one of repair in the presence of a granulation tissue.

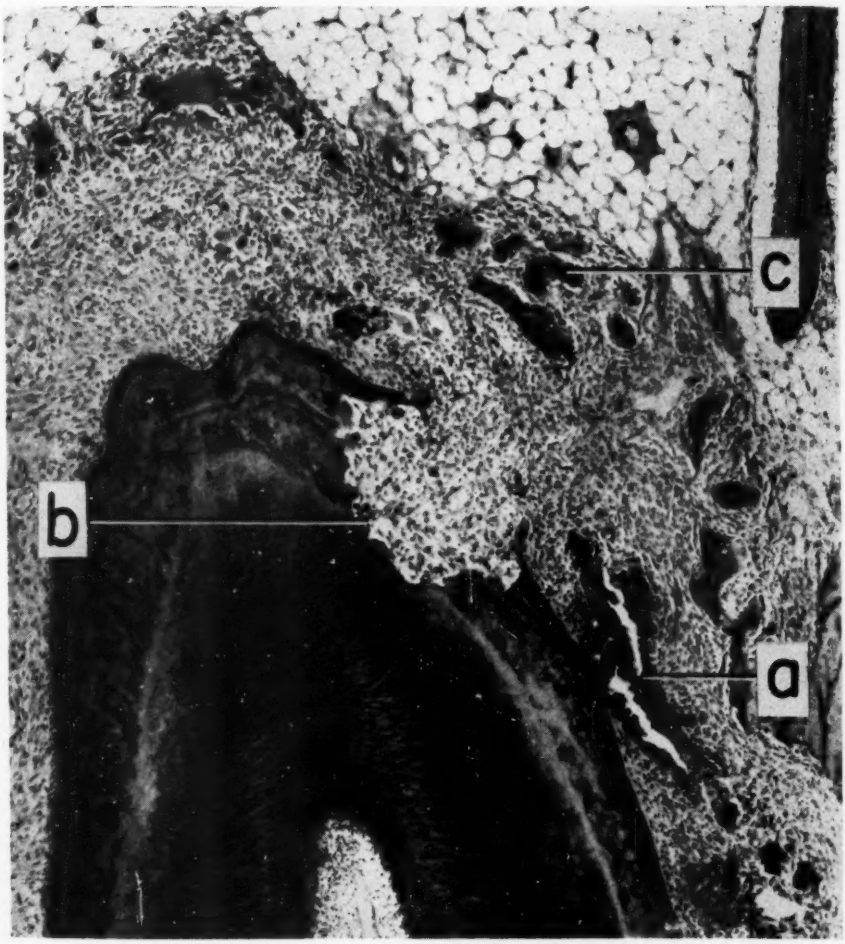

Fig. 7. (TM-1) Fourteen day experiment. Palatal root. Extensive necrosis has occurred on the palatal surface of this root (a) but resorption has almost eliminated the area of compression. There is resorption on the root surface (b) and the alveolar bone (lamina dura) is broken up into small islands of bone (c) with resorption in progress in some regions and new bone formation on others.

TWENTY-ONE DAY ANIMAL

Histologically in this experiment all the signs of tissue damage and progressing repair are still in evidence (Fig. 8 A-B).

A and B of Fig. 8 are different sections of the same series. One shows more of the mesio buccal root, the other more the disto buccal and the palatal root.

Buccal Side: Fig. 9 shows a high magnification of the buccal alveolar crest. Osteoclasts have produced lacunae in the bone tissue and cementum facing the periodontal ligament. This resorption has caused a widening of the periodontal space, which now measures an average of .32 mm. as compared with the 3 day animal average of .19 mm.

Fig. 8A. (TM4-1) Three weeks experiment. Mesio-buccal and palatal roots a—buccal alveolar crest. b—lingual alveolar crest, c—resorption of root surface close to the apex of the buccal root, d—compression in the bifurcation.

Fig. 8B. (TM4-1) Three weeks experiment. The same tooth as seen in Fig. 8A—but more distal in the serial sections, showing the disto-buccal root and palatal root. a — buccal alveolar crest, b — lingual alveolar crest, c and d pressure areas in the bifurcation, e — pressure area at the apex of the palatal root.

The Figures 8A and B show the extensive traumatic tissue changes in different areas of the same tooth — to clarify the third dimensional aspect.

Some new bone has been laid down on the inside of the alveolar socket of this tooth. In addition the periodontal ligament shows some widened capillaries. Closer to the apex of this same root on the buccal side a pressure area and osteoclastic action may be observed (Fig. 10).

Palatal Area: This area is comparable to the buccal area. Here also are signs of compression and resulting widening of the periodontal ligament. Resorption of bone and cementum is the predominant picture, and this in turn due to osteoclastic activity has led to widening of the periodontal space (Figs. 11, 12). Figures 11 and 12 are different sections of the same tooth. 11 corresponds to Fig. 8A, 12 corresponds to 8B, showing that in one area compression with undermining resorption is in progress—in the other the widening has reached a stage where pressure has been eliminated.

Bifurcation Area: Extensive necrosis followed by bone resorption has occurred on both the palatal and buccal surfaces of the inter-radicular septum (Fig. 13).

Apical Area: Necrosis has also occurred in the apical region of this tooth (Fig. 14). Resorption of bone and cementum is in progress. Endosteal bone formation is occurring on the marrow side opposite the sites of bone resorption.

THREE MONTH ANIMAL

In this animal as in the previous one, the experimental tooth was very loose. Clinical examination did not show any difference in the texture or coloration of the gingiva on the experimental or control sides. The microscopic findings were as follows:

Three months after the onset of the experimental procedure, no active traumatic tissue changes could be observed in any portion of the periodontal ligament (Fig. 15).

A characteristic feature of the three month experiment was the lengthening of the periodontal ligament and the widening

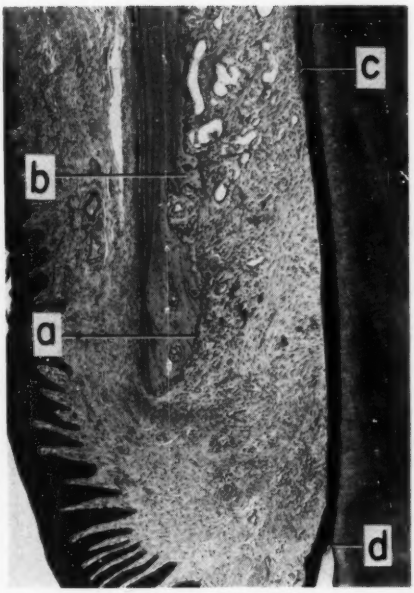

Fig. 9. (TM4-1) Three weeks experiment. Buccal alveolar crest—higher magnification of area (a) of Fig. 8A. The periodontal space is considerably widened. Resorption is active on the cemental surface (c). Slight resorption is also active at the tip of the alveolar crest (a), while further apical new bone is laid down on the periodontal side (b) as well as on the periosteal side of the crest. d — cemento-enamel junction.

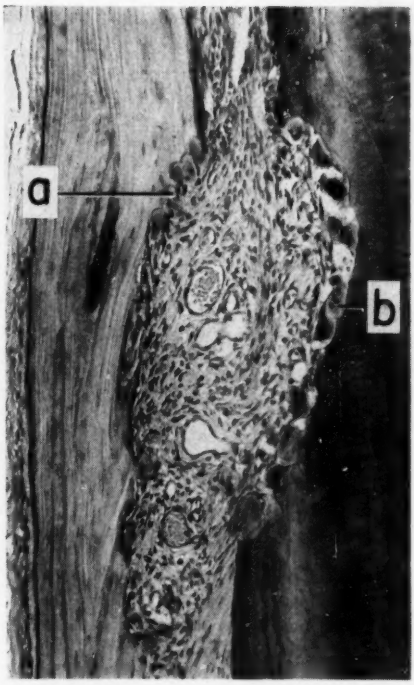

Fig. 10. (TM4-1) Three weeks experiment. Higher magnification of area c from Fig. 8A. Pressure area near apex showing active resorption of tooth (a) as well as bone (b).

of the periodontal space (Width average .65 mm.). Compare this with the three day experimental animal (Fig. 2), where the width of the periodontal space measured average .19 mm.

Buccal Side: High magnification exhibits the extensive widening of the periodontal space. Osteoclastic and more extensive osteo-blastic activity are still in progress (Fig. 16). New bone has been laid down on the buccal, periosteal surface of the alveolar process.

Palatal Side: Shows the same characteristic widening of the periodontal space.

Apical Area: In this region there is an extensive widening of the periodontal space. There is no apparent tissue damage. The connective tissue appears more cellular but no necrosis or thrombosis is observable (Fig. 17).

Bifurcation Area: A slight resorption is still occurring. A small area of necrosis is apparent in one spot.

SIX MONTH ANIMAL

Six months after the cementing of the crown and the palatal appliance, the tooth was still extremely loose. No changes could be observed in the gingiva of either the experimental or control tooth. The bottom of the sulcus was still on the enamel; there was no pocket formation.

The microscopic changes in the tooth and its supporting structures at six months post operatively were similar to those observed at three months.

The characteristic widening of the periodontal space may be observed in Fig. 18. Areas of repaired resorption are evident on

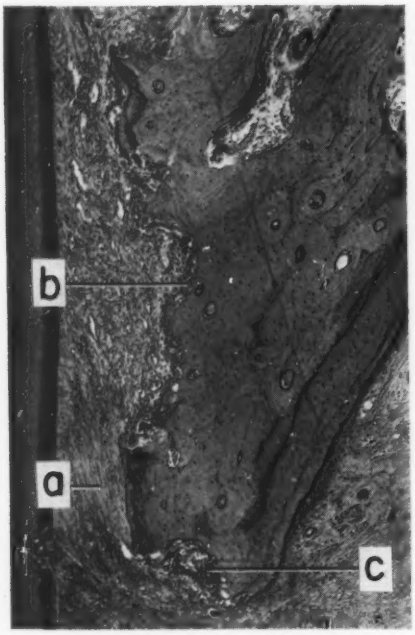

Fig. 11. (TM4-1) Three weeks experiment. Higher magnification of the lingual alveolar crest, area b from Fig. 8A. Compression of periodontal ligament at a — and active undermining bone resorption at b and c. There is a small resorption area also on the tooth—coronal of the pressure area.

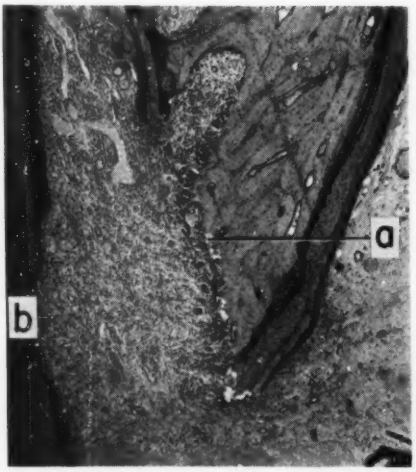

Fig. 12. (TM4-1) Three weeks experiment. Lingual alveolar crest of section shown in Fig. 8B. Widening of the periodontal space by resorption of the alveolar bone (a) and also resorption of the tooth (b).

the palatal side of the buccal root close to the apex and at the center area of the palatal surface of the palatal root (Fig 18). No signs of necrosis, or thrombosis are present. It is evident that no alteration in the position of the epithelial attachment has occurred if checked and compared to the position of the epithelial attachment of the control tooth.

DISCUSSION

I. Histologic changes in excessive occlusal stresses: In a number of previous investigations 1, 2, 3, 4, 5, it has been shown that excessive occlusal stresses lead to thrombosis and hemorrhage, destruction of bone and necrosis of collagen fibers on the areas where pressure is exerted. On the tension side, over extension of the unellastic collagenous fiber bundles occurred with hemorrhage and thrombosis of blood vessels accompanied by resorption of bone as well as cementum.[2] However, in these "orthodontic type" traumatic experiments the tooth escaped the trauma and on the side of tension new bone formation occurred. Finally complete regeneration of the damaged periodontium occurred on both the pressure side and the tension side.

In the present study the experimental tooth was not allowed to escape the trauma and adjust in this manner by moving out of range of the excessive forces. The trauma was exerted constantly whenever the animal brought his teeth into function at any period during the entire six month experiment. This means also that the periodontium did not reveal as in the previous studies a definite pressure side or a definite tension side, rather on all traumatized areas the effects of *both* tension and pressure were recorded as the tooth was "jiggled" in a buccal and then palatal direction. The combined effect of pressure and then tension were recorded as thrombosis of blood vessels, necrosis of the periodontal ligament followed by resorption of bone and cementum. As a consequence of the continued trauma, the resorption of bone and necrotic

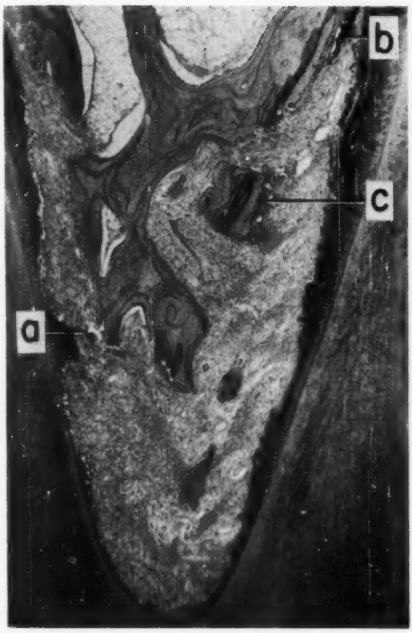

Fig. 13. (TM4-1) Three weeks experiment. Bifurcation of tooth shown in Fig. 8B, showing pressure areas on the buccal (a) as well as palatal (b) side of the inter-radicular septum, with active resorption of bone (c) as well as cementum going on.

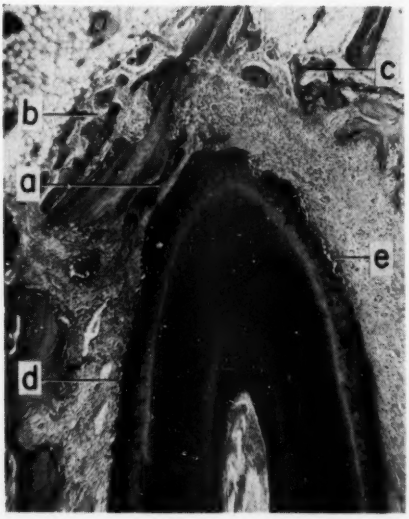

Fig. 14. (TM4-1) Three weeks experiment. Apex of the palatal root of the tooth shown in Fig. 8B. (e). There is a pressure necrosis area (a) with multiple small resorptions of the cementum on the buccal (d) as well as palatal (e) surface. Osteophytic (immature) type of bone formation on the endosteal surfaces of bone trabeculae (b and c).

connective tissue led to an increasing widening of the periodontal space until it was more than three times as wide as a none traumatized tooth. But at one point, the damaging effect of the jiggling trauma was nullified by the extreme width of the periodontal space and no further crush necrosis occurred. Now, the periodontal ligament regenerated, the bone tissue healed, the cementum showed repair. The tooth and its periodontium may be said to have adjusted to increase functional demands. An additional functional compensatory tissue change took place in the form of new bone formation on the periosteal and endosteal surfaces of the bone involved (Figs. 4, 6, 14, 16).

Clinically the tooth appeared to be extremely loose. It may be stated that the firmness of the tooth in its socket depends

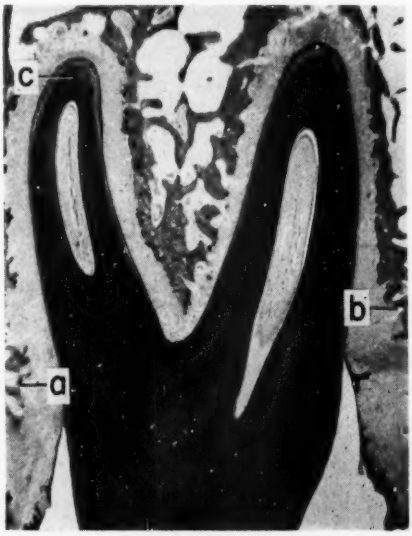

Fig. 15. (TM3-1) Three months experiment. Note the width of the periodontal space (average .65 mm.). Compare this with the width of the periodontal space of the tooth in a 3 day experiment (Fig. 2 average .19 mm.).

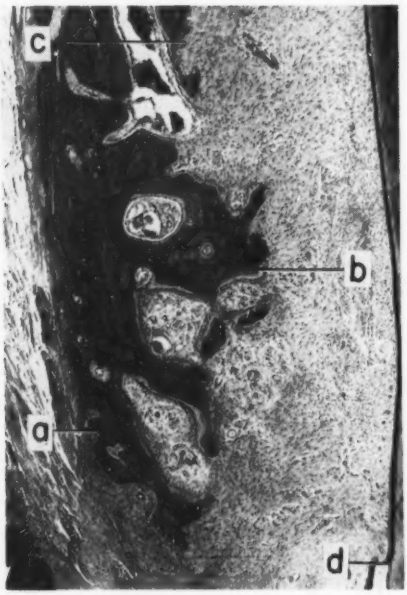

Fig. 16. (TM3-1) Three months experiment. Higher magnification of the buccal alveolar crest. Bone formation on the periosteal (a) as well as periodontal surface (b). Limited area of bone resorption (c). Cemento-enamel junction (d).

Fig. 17. (TM3-1) Three months experiment. Apex of the buccal root (Fig. 15c). Wide periodontal space. The fiber bundles in some areas are well organized, in others the connective tissue is loosely arranged with increased width and number of capillaries.

Fig. 18. (TM2-3) Six months experiment. Periodontal space widened. No acute signs of traumatic tissue changes are seen! Repaired root resorptions are on both roots (b and c) a buccal alveolar crest.

upon two factors, if root length is a constant:

1. The length of the periodontal ligament (width of periodontal space).

2. The height of the alveolar crest.

In this experiment the lengthening of the periodontal ligament and therefore widening of the periodontal space must be regarded as a tissue adaptation to increased functional demands. One consequence of the latter is increased mobility of the experimental tooth in its socket. This looseness is not the sign of an inflammatory periodontal disease state as there is no gingivitis, no pockets, no down growth of the epithelial attachment. This is a functional readjustment of normal tissues to continued occlusal stress.

II. Occlusal trauma and periodontal disease:

The question may still remain however, of what consequence would a previous gin-

gival and periodontal inflammation have in the presence of the damaged tissues caused by continued occlusal trauma. From our experience (Weinmann and Macapanpan)[6] we may venture to assert that inflammation and infection would alter the traumatic tissue changes or vice versa, the traumatic tissue changes may possibly influence the severity of a previously existing periodontitis. This must be the basis of new animal experiments i.e. to introduce *"jiggling" tissue trauma* in the presence of a previous inflammation of the periodontium.

Here in a new experimental series may be the answer to the question, "What is the *combined effect* of 1. *continued jiggling tissue trauma* and 2. periodontal inflammation upon the severity of periodontal disease?

SUMMARY

The effects of jiggling occlusal trauma upon the periodontium were experimentally studied in 6 monkeys. Crowns with lingual locks were cemented to the maxillary second premolars on the right side. The crowns were fabricated in such manner that the teeth were subjected to excessive occlusal stresses in the buccal direction at each occlusal closure. A palatal orthodontic appliance with finger spring was connected to position to the second molars. The finger spring when engaged into the lingual tooth of the crown—exerted a pull in lingual direction when the teeth were disoccluded. This arrangement produced a bucco-lingual jiggling trauma. The experiments lasted 3 days, 2 weeks, 3 weeks, 3 months and 6 months.

CONCLUSIONS

The early transient tissue changes in the 3 days to 3 weeks experiments were identical to those described in previous traumatic occlusion experiments—except that pressure and tension areas occurred on both the buccal and lingual areas. The end results of this experimental arrangement differed from previous experiments since the traumatized teeth could not escape the trauma by migration—but showed in 3 months and 6 months experiments a compensatory elongation of the periodontal ligament—with widening of the periodontal space to more than three times the original width.

Traumatic tissue changes of the early experiments completely disappeared within 3 months and the periodontium appeared normal except for the lengthened periodontal ligament. The widening of the periodontal space and lengthening of the periodontal ligament may be considered as a functional adaptation of the tissues due to changes in the functional requirements.

No gingivitis or periodontitis was evident in either the 3 months or 6 months experiments.

REFERENCES

1. Gottlieb, B. and Orban, B. Die Veranderungen der Gewebe bei ubermassiger Beanspruchung der Zahne Thieme—Leipzig, 1931.

2. Bhaskar, S. and Orban, B. Experimental Occlusal Trauma. J. Period. 26:270, 1955.

3. Orban, B. Tissue Changes in Traumatic Occlusion. J.A.D.A. 15:2090, 1928.

4. Grohs, R. Veranderungen in Periodontium bei uberlasteten menschlichen Zahne. Z. Stomatol. 29:386, 1931. J. Dent. Res. 11:510, 1931.

5. Orban, B. Traumatic Occlusion and Gum Inflammation. J. of Period. 10:39, 1939.

6. Macapanpan, Luz C. and Weinmann, J. P. The Influence of Injury to the Periodontal Membrane on the Spread of Gingival Inflammation. J. Dent. Research 33:263, 1954.

A Logical Approach to Periodontal Pocket Therapy

by James Howells, B.S., D.D.S.,* Herbert I. Oshrain, B.S., D.D.S.,* Clinton D. Rehnke, D.D.S.,* Albert Salkind, D.D.S.,* William Shoulberg, D.D.S.,* Joe M. Smith, B.S., D.D.S.,* Ray M. Spyres, A.B., D.D.S.*

THE divergence of opinion concerning the complete elimination or retention of periodontal pockets after periodontal disease has been treated, has caused much confusion, and practitioners are uncertain as to the correct approach to the selection of therapeutic procedures. Criteria should be established for the treatment of periodontal pockets. On the basis of this criteria, it can then be determined when to completely eliminate pockets or when they should be allowed to remain. By doing so, it can then be determined when a case is a successfully treated one, or one that should be considered a compromise. Therapy rendered in this manner is both scientific and efficient.

The rationale for an approach to therapy is contingent on the word "cure." Since cure is the return to health, it would seem logical to examine the lesion analytically and decide when the lesion no longer exists and the periodontal tissues have returned to health.

A periodontal pocket may be defined as a gingival sulcus pathologically deepened by periodontal disease.[1] The usual clinical signs and symptoms of pocket formation are increased sulcular depth (either apically or occlusally), discoloration, loss of stippling, bleeding, presence of an exudate, and the distortion of architectural form. Histologically, there may be ulceration of the pocket epithelium, apical downgrowth of the epithelial attachment, inflammatory infiltrate among the connective tissue fibers, destructive changes in the connective tissue, and resorptive changes in the bone. Radiographically, these resorptive changes are apparent as osseous architectural deformities. Unfortunately, as clinicians we have only the clinical and radiographic evidence to rely upon.

The very nature of the pocket serves as a haven for calculus, bacteria, food and tissue debris. These noxious agents consistently irritate periodontal tissues to cause the formation of the pockets in majority of cases. One must emphasize that the apical migration of epithelium is the result of an initial breakdown of periodontal connective tissue fibers or an initial proliferation of the epithelium. It is the inflammatory process which sets off the reaction.[2] Investigations of Aisenberg and Aisenberg, Duran-Reynals, Engel, Gersh and Catchpole, and Schultz-Haudt, Bibby and Bruce[3-7] have suggested that bacterial growth produces an enzyme, hyaluronidase-like in nature, which functions as a "spreading factor." These investigations strongly entertain the possibility that the hyaluronic acid content of the connective tissue is depolymerised by the action of hyaluronidase, breaking down the connective tissue barrier allowing for the apical proliferation of the epithelial attachment.

Accordingly, a return to health should involve the following criteria:

a. The elimination of all irritants.

b. The resolution of inflammation and eradication of all its sequelae, particularly the periodontal pocket.

c. The restoration of proper form of the gingivae and bone so that a non-destructive climate can be achieved in the functioning of the teeth and supporting tissues.

There is no general agreement among authorities in the field of periodontology as to what constitutes cure for the periodontal lesion. The following authorities have given their views on this subject in "In Our Opinion."[8] Leonard says, "Tissues

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will be maintained in health with no signs of further deterioration even though sulci* of considerable depth remain demonstrable." I. Hirschfeld states, "After periodontal pockets have been successfully treated, the remaining healthy gingival flap forming the pocket may continue in health indefinitely even though complete reattachment is not obtained. In other words, the retention of such completely treated tissue is not necessarily conducive to reinfection if oral hygiene and function are maintained to a reasonable degree. This statement is made on the basis of periodontal examinations of thousands of formerly involved teeth over a period of many years." Lyons concludes, "Good health can and does prevail in sulci* with marked depth." However, Beube asserts, "The presence of measurable pockets with healthy gingival tissues is not an indication of normalcy. There is a constant inflammatory process at the base of these pockets." Goldman states, "It is the aim of periodontal therapy not only for the gingivae to heal, resulting in a normal gingival sulcus, but also for the contour to be physiologically within the norm for the particular area." Ramfjord claims, "The ideal way to eliminate the pocket is by reattachment following subgingival curettage, but only in a relatively limited percentage of cases can a situation be established which is conducive to reattachment."

Arnim and Hagerman, Beube, Morris, Shaffer and Zander,[9-12] in independent experimentation have demonstrated clinically and histologically that tissues can be restored to health by thorough curettage. It is apparent that the periodontal pocket and the accompanying inflammation can be eradicated by means of subgingival curettage or surgery or by both methods. Should subgingival curettage precede surgery, the pocket may be eliminated by the loss of edema with subsequent shrinkage, or by reattachment. Periodontal surgery should follow curettage in order to eliminate any residual pockets. This may be accomplished by excision or by reattachment.

The question then resolves itself as to the concept of pathosis. Bell[13] defines pathology as the study of the causes of disease and the structural, chemical, and functional alterations in the body resulting from disease. If, in the minds of those who contend that periodontal tissues can be maintained in the presence of periodontal pockets, why then is it also necessary to remove the deformity, namely the periodontal pocket? In line with the previous definition of pathology it would appear as though no cure has been effected until all the pathologic entities have been removed. This involves the return of tissue tone and proper form. Followers of the school of correct form and function feel that tissue can be altered so that as many of the aggravating factors as possible are eliminated. According to Goldman, Schluger, and Fox,[14] "One of the primary objectives of periodontal therapy is the eradication or arrest of the periodontal lesion with the correction or cure of the deformity created by it."

The treatment of pockets by surgical elimination or by subgingival curettage may create new problems. The new environment of the gingival margin must be considered. The margin, having been placed in a new position may be susceptible to trauma and food impaction. Therefore, in any attempt to eradicate a pocket the recreation of physiologic architectural form must be realized to insure healthy gingival tissues. Such an environment is secured by reshaping teeth so that deflecting contours are created, and by beveling and festooning periodontal tissues to allow for physiologic function. With gingival margins so protected, accumulation of food is prevented, trauma is reduced, and the patient can practice oral physiotherapy more effectively.

If one's philosophy of treatment is the elimination of etiologic factors and pathologic changes, then to cease treatment while pockets exist merely because there is no

*The authors believe that a sulcus which has been deepened is a pocket, even if the tissues appear to be clinically healthy.

clinical sign of inflammation should be considered incomplete therapy.

Providing the theory of complete pocket eradication can be accepted as the approach to the ideal, we must still recognize certain compromising circumstances. The first indication for compromise is the systemic condition of the patient. Age must be considered. The aged and infirm may not react well to the trauma of surgery and this phase of treatment may have to be omitted.[15] Factors such as blood dyscrasias, circulatory disorders, metabolic and degenerative diseases may affect the mode of treatment. In some instances, treatment must be minimized. Such a case is the hemophiliac. In this disorder, the individual cannot undergo surgery, but can be treated with subgingival curettage provided patient is hospitalized and proper precautions are taken. Another situation warranting compromise is the psychological state of the patient. There are individuals who are psychogenically traumatized by the extraction of a tooth. To such patients this is comparable to an amputation. The same reaction expresses itself in patients subjected to the excision of gingiva or bone. It should be possible to elicit this amputation complex as well as other systemic and psychologic factors with the taking of a careful history. Other indications for compromise are to be found in areas where the elimination of pockets is not feasible. Examples are the inherent nature of the area, the location of the pockets, the degree of destruction, and the esthetic appearance.

The logical sequence of treatment of the periodontal pocket is to eliminate all irritants by subgingival curettage in an effort to obtain shrinkage, resolve inflammation, and possibly achieve reattachment. Any deformity that remains should be removed by periodontal surgery.

By following such a procedure, periodontal therapy no longer has to fit into a pattern of trial and error. A standard of certainty and assurance is attained, a healthy periodontium can be effected and a greater number of teeth will be saved.

The authors wish to express their appreciation for the advice and constructive criticism of Dr. Frank E. Beube, Clinical Professor of Dentistry and Head of the Section of Periodontology, School of Dental and Oral Surgery of the Faculty of Medicine, Columbia University.

BIBLIOGRAPHY

1. Glickman, I.: Clinical Periodontology, W. B. Saunders Co., Philadelphia 1953, p. 233.

2. Goldman, H.: Periodontia, C. V. Mosby Co., St. Louis, 1953, 3rd Edition, p. 75.

3. Aisenberg, M. S. and Aisenberg, A. D.: Hyaluronidase in Periodontal Disease. Oral Surgery, Oral Medicine & Oral Pathology. 4:317 1951.

4. Duran-Reynals, F.: Tissue permeability and the spreading factors in infection, a contribution to the host parasite problem. Bact. Rev. 6:197 1942.

5. Engel, M. B.: Some recent concepts of the connective tissue ground substance and their implications in dental disease. J. Periodont. 21:145 1950.

6. Gersh, I. and Catchpole, H. R.: Organization of ground substance and basement membrane and its significance in tissue injury disease and growth. Am. J. Anat. 85:457 1949.

7. Schultz-Haudt, S., Bibby, B. G., and Bruce, M. A.: Tissue destructive products of gingival bacteria from non-specific gingivitis. J. Dent. Res. 33:624 1954.

8. Leonard, H. J. et al: Is reattachment or pocket elimination essential for retaining periodontally affected teeth in health? J. Periodont. 19:21 1948.

9. Hagerman, D. A., and Arnim, S. S.: The effect of periodontal treatment on the connective tissue fibers of the marginal gingivae. J.A.D.A. 48:158 1954.

10. Beube, F. E.: A study on reattachment of the supporting structures of the teeth. J. Periodont. 18:55 1947.

11. Morris, M.: Healing of naturally occurring periodontal pockets about vital human teeth. J. Periodont. 26:285 1955.

12. Shaffer, E. M. and Zander, H. A.: Histologic evidence of periodontal reattachment of pockets. Paradent. 7:101 1953.

13. Bell, E. T.: Textbook of Pathology, Lea & Febiger, Philadelphia 1952, p. 19.

14. Goldman, H. M., Schluger, S., and Fox, L.: Periodontal Therapy, C. V. Mosby Co., St. Louis 1956, p. 53.

15. Beube, F. E.: Periodontology, Macmillan Co. New York 1953, p. 518.

Periodontal Prosthesis in Occlusal Trauma

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SEVERAL investigators have performed experiments on animals, creating traumatic occlusal stresses on individual teeth in order to determine the relation of occlusal trauma to the apical migration of the epithelial attachment, i. e. periodontal pocket formation.[1,2,3] In the experimental animals used there was no increase in depth of the pocket unless local irritants such as calculus were added.[4]

Sternlicht[5] reported a clinical case of primary periodontal traumatism treated in cooperation with Ochsenbein, in which trauma was caused by removable partial dentures, constructed after successful periodontal therapy. Periodontal pockets did not recur when the teeth were subjected to severe trauma for a period of fourteen months and the traumatic lesions evidenced by the widening of the periodontal membrane spaces healed rapidly following stabilization with fixed periodontal prosthesis.

The purpose of this report is to present two clinical cases of primary periodontal traumatism without other periodontal complications such as the presence of calculus and deepened sulci, with successful therapy by means of fixed periodontal prosthesis.

The role of occlusal trauma in the etiology of periodontal disease has caused great confusion, but clinical and histologic evidence both clearly show the inflammatory and traumatic lesions to be separate entities. The periodontal traumatic lesion is noninflammatory and it is localized in the attachment apparatus and does not influence the gingival fibers.[6,7,8,9,10,11,12,13] No theoretical explanation for periodontal pocket formation due to trauma has survived scientific investigation. Orban states that inflammatory and traumatic lesions are both present in possibly 90 per cent of all patients, which accounts for the common finding of inflammatory and traumatic changes in the same areas and at the same time.

Periodontal traumatism is the result of occlusal trauma and is defined in Blakiston's New Gould Medical Dictionary, Second Edition, as "Injury of periodontium due to excessive occlusal, operative, accidental, or orthodontic stress." Mal occlusion does not necessarily cause occlusal trauma, although occlusal imbalances in initial occlusal contact may act as a trigger mechanism, causing habits which produce periodontal traumatism.[14] Damage to the attachment apparatus does not occur during normal function, that is, mastication, swallowing, and talking. Teeth seldom contact during mastication and forces exerted on them are small—8 to 10 pounds—with pressure building up gradually, never being delivered as a blow.[15,16,17] These forces are predominantly vertical in direction while tensional habits of occlusal neurosis, such as bruxism and clamping introduce horizontal component of force.[18,19,20] In tooth to tooth contact, such as gritting, clamping, or clenching, forces approaching an individual's body weight may be exerted, creating damage to the periodontium.

When bone absorption has progressed to such an extent that the clinical crown—clinical root ratio has become an unfavorable one, secondary occlusal trauma occurs during function. Transitory instances of periodontal traumatism will be followed by repair within twenty-four hours and permanent damage does not result. The magnitude, direction, distribution, and frequency of the force and the repairative capacity of the individual determine the extent of the lesion.[20,21]

Diagnosis of periodontal traumatism is made by clinical and roentgenographic examination. Clinical evidence of trauma to the periodontium is seen in abnormal mo-

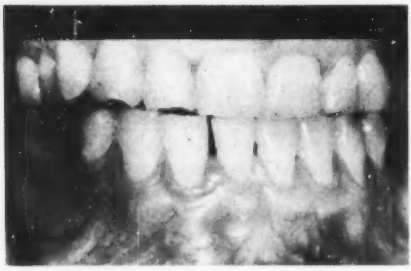

Fig. 1, Case 1: Photograph showing abrasion of the right maxillary lateral incisor and cuspid with facets on the mandibular cuspid and first premolar. The abraded areas occlude beyond the range of functional movement. The right mandibular lateral incisor has moved out of contact with the right central incisor and cuspid and the marginal gingiva of the lateral incisor is enlarged due to loss of contact. Sulcus depth is almost ideal.

bility of the tooth or teeth involved. Teeth do not loosen from marginal periodontitis alone until the supporting bone has absorbed to a point near the apex of the root and then it is usually on more than one root surface. Afunctional wear patterns and shiney facets are observed as indications of habit patterns. Tension and tenderness in the muscles of mastication as determined by palpation are indicative of isometric contraction which is the motor outlet to emotional strain.[22]

Roentgenographic evidence of periodontal traumatism is seen as a widening of the periodontal membrane space, absorption of alveolar bone (lamina dura) and root absorption.[23, 24, 25] An enlarged periodontal membrane space has also been observed in some cases of acrosclerosis and diffuse scleroderma, systemic diseases affecting collagen.[26, 27] It is possible that occlusal stress may be responsible for the enlarged periodontal membrane space here, the collagen disease preventing adequate repair.

The histologic appearance of the periodontal traumatic lesion has been described as one of hemorrhage, thrombosis of blood vessels, cemental tears, necrosis, and bone and cementum absorption. Hyalinization of the connective tissue of the periodontal membrane and cartilage formation may occur in cases of long standing.[2, 6, 9]

The periodontal traumatic lesion may be treated by[1] selective grinding,[2] orthodontics, or[3] periodontal prosthesis or by a combination of the above. All grinding procedures are designed to narrow occlusal tables, remove lateral interferences, and to distribute forces over groups of teeth to bring stresses to a point which the *patient can afford in terms of bone.* If a tooth is found to be loose, its range of function should be reduced by narrowing the crowns bucco-lingually and grasping contacts should be relieved by reduction of steep incline planes; if this proves inadequate, splinting should be applied. Dorland's American Medical Dictionary, Twentieth Edition, defines a splint as "A rigid or flexible appliance for the fixation of displaced or movable parts." If unusual stress is expected because of occlusal habits of clenching or gritting, palliative appliances such as plastic night splints are constructed. If a tooth has moved out of contact or into

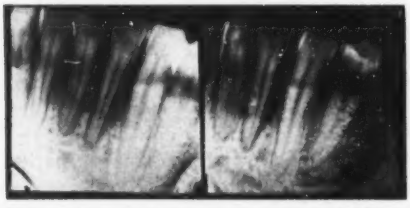

Fig. 2 A, Case 1: Roentgenograms, 6/3/54, of mandibular incisors showing enlarged periodontal membrane spaces with alveolar bone and root absorption due to severe and prolonged occlusal trauma. All roentgenograms are seen from the lingual side. Left: Projection through embrasure between the right central and lateral incisors. Right: Projection through embrasure between the right lateral incisor and cuspid.

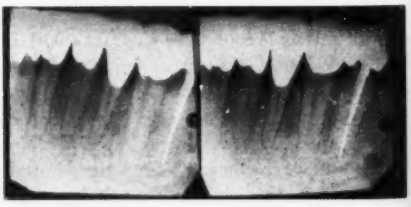

Fig. 2 B: Roentgenograms of the same regions showing the regeneration of alveolar bone with narrowing of the periodontal membrane spaces 14 months after fixation with periodontal prosthesis.

too heavy contact, it should be moved by orthodontic therapy.

Therapy for the cases reported here was stabilization of loose teeth with fixed periodontal prosthesis or splints. Splinting changes the center and axis of rotation, creating one multi-rooted tooth by the appliance. The established plane of occlusion permits the mandible to move freely in any direction without interference. A centric occlusal pattern was created in the occluding surfaces which was definite enough to orient the mandible comfortably, but not so deep as to create torque in any excursion from it.[28]

Full coverage castings were used, not only because of the strength and retention offered, but also in order to effect a complete reorientation of the occlusion for distribution of stress and of tooth anatomy for protection of the gingiva. The prosthetic material was terminated at the gingival margin or coronal to it. This is important in the use of multiple abutments as inflammation and marginal thickening can usually be avoided by terminating the crowns near the gingival margin, but not beneath it. Tissue trauma from pressure packs or cauterization in preparation for impressions beneath the gingiva were also carefully avoided.

The splints may be retained with temporary cement and can be removed for inspection of individual teeth. In both cases below, the extremely mobile teeth were firm in their sockets six months after completion of the splint.

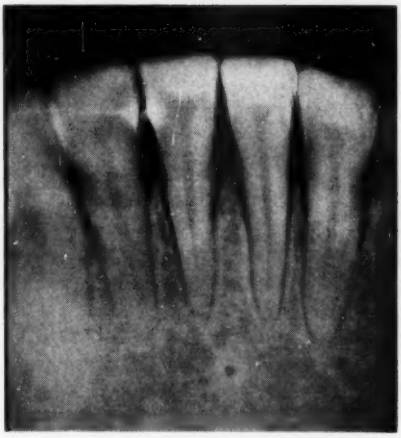

Fig. 3, Case 1: Roentgenogram of mandibular incisors made on January 17, 1949 showing enlarged periodontal membrane spaces indicative of occlusal trauma.

CASE #1

A 29 year old housewife was examined on June 3, 1954. Her chief complaint was that she could not wear a removable partial denture replacing the missing mandibular posterior teeth because of vague diffuse pain. She was also concerned because her teeth were loose. The patient appeared to be an extremely nervous person. She had been married twice, having had three children by her first husband and she was now in the fifth month of pregnancy.

Her past dental history included the occasional extraction of posterior teeth over a period of several

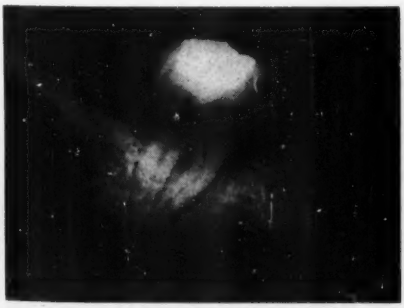

Fig. 4, Case 1: Roentgenogram of left mandibular molar made on December 8, 1947 showing enlarged periodontal membrane space and root absorption due to severe occlusal trauma.

Fig. 5, Case 1: Photograph of periodontal prosthesis 14 months after completion. The marginal gingiva of the right lateral incisor is still enlarged, and sulcus depth has not changed.

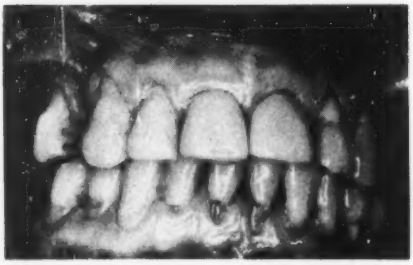

Fig. 6, Case 2: Photograph before splinting. All of the teeth are mobile, ranging from moderate to severe. Gingival sulcus depth is within normal range.

years. All mandibular molars had been removed and on the right side, the second premolar was also missing. In the maxillary arch all teeth were present except the third molars and the left second molar.

Gingival sulcus depth throughout the mouth approached the ideal, and there was evidence of good oral hygiene. All of the mandibular teeth exhibited abnormal mobility. The right central and lateral incisors felt to the examining fingers as if they were imbedded in soft rubber; the lateral incisor was more mobile than the central incisor. These teeth could be elevated and slightly depressed in their sockets and could be moved freely in a labio-lingual direction. Mesio-distal movement was limited only by the adjoining teeth. The marginal gingiva on the mesial of the lateral incisor was enlarged and slightly inflammed due to the loss of contact and irritation from stretching with the extreme labial movement. There were deep notches in the incisal surfaces of the maxillary right cuspid and lateral incisor with lingual cupping in the dentine (Fig. 1). The opposing mandibular teeth occluded with these worn areas beyond the range of functional movement.

The patient was not conscious of occlusal habits other than jiggling the loose lower incisors when they were sore. The muscles of mastication were tense and palpation of the left masseter muscle caused acute pain.

Roentgenograms of the mandibular incisors (Fig. 2 A) show enlarged periodontal mebrane spaces with bone and cementum absorption, indicative of prolonged and severe periodontal traumatism. Figure 3 shows a roentgenogram of the same area that was made in 1949, and Figure 4 shows a pre-extraction roentgenogram of a mandibular molar made in 1947. Roentgenographic evidence of trauma is seen in these illustrations.

Immediate therapy consisted of infiltration of a local anesthetic into the trigger area of the left masseter muscle.[29] This relieved the muscle spasm which has not recurred. Fixed splinting of the remaining mandibular teeth was recommended for stabilization, but was not possible for economic reasons. The mandibular cuspids and incisors were ligated with stainless steel ligature which was then covered with a self-curing acrylic resin on June 11, 1954.[20, 21, 22] An acrylic night guard was also constructed, but could not be adequately stabilized on the few remaining teeth, so it was discarded. Pregnancy was terminated by uneventful delivery on September 27, 1954.

The temporary splinting as described, provided inadequate stabilization as the teeth and splint moved in unison, however, it did limit the extreme movement of the right incisors. It was observed for a year during which time oral hygiene became difficult because food debris was retained by the acrylic and gingival irritation developed. Also, the right mandibular first premolar became painful to pressure.

By August of 1955 it was evident that the remaining mandibular teeth must either be stabilized with fixed splints or be extracted. The patient's husband was then unemployed, but fixed splinting was made available to the patient, which she accepted. Figure 2 B, shows the roentgenographic evi-

Fig. 7, Case 2: Roentgenograms showing enlarged periodontal membrane spaces and root absorption due to severe and prolonged occlusal trauma.

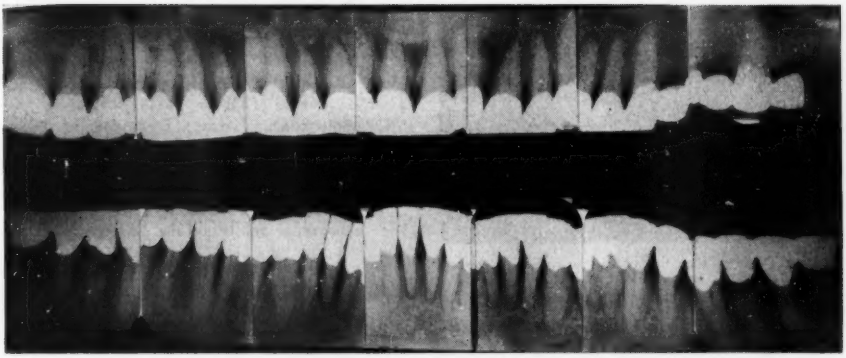

Fig. 8, Case 2: Roentgenograms with castings in position during construction of splints.

dence of healing fourteen months after final splinting and Figure 5 shows the clinical appearance at that time. The thickened gingival margin is noticeable on the mesial of the right lateral incisor, but the sulcus did not become deeper even though some irritation developed during the year of temporary splinting. The right mandibular first premolar was treated endodontically to relieve traumatic pulpitis. A removable partial denture was not made to replace the missing mandibular molars. The patient could chew adequately, and we wished to avoid the additional torque that a removable appliance would create.

CASE #2

A 48 year old housewife's chief complaint was discomfort during mastication because of loose teeth. She thought that she could "remove her teeth with her fingers." She was aware of occlusal habits, primarily of severe and prolonged clamping. Three consultants had recommended complete extraction.

On examination it was found that all of the teeth were mobile ranging from moderate to severe. The mandibular incisors and premolars could be elevated and depressed in their sockets. All of the mandibular molars were missing, also the right maxillary molars and first premolar. The left maxillary teeth were all present except the second and third molars. The missing teeth had been replaced with removable partial dentures. Gingival sulcus depth was within normal range. Clinical appearance at the time of examination is seen in Figure 6. Oral roentgenograms gave evidence of periodontal traumatism (Figs. 7 and 8). The muscles of mastication were tense and palpation

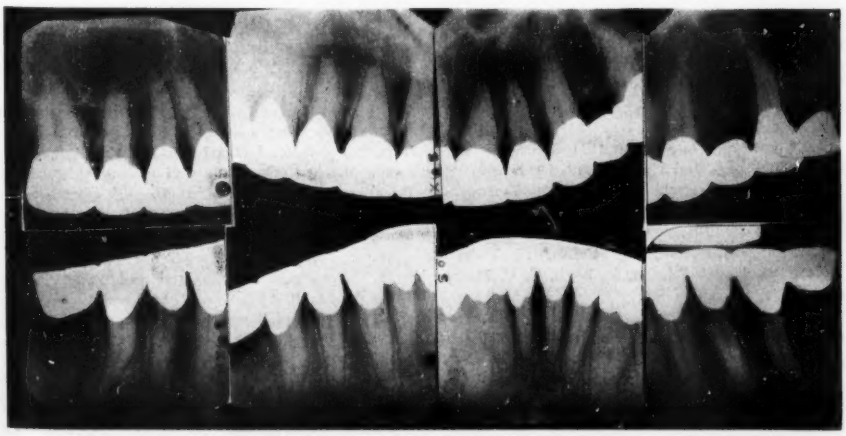

Fig. 9, Case 2: Roentgenograms showing narrowing of periodontal membrane spaces 12 months after fixation with periodontal prosthesis.

Fig. 10, Case 2: Photograph of periodontal prosthesis and gingivae 12 months after completion.

caused pain, but there were no trigger areas of acute sensitivity. Figure 9 shows the roentgenograms one year after the final splints were completed and Figure 10 shows the clinical appearance of the prosthesis at that time.

SUMMARY

Two cases of primary periodontal traumatism without periodontal pocket formation have been presented. Therapy, by stabilization with fixed splints, resulted in healing of the damaged attachment apparatus as observed clinically and roentgenographically.

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REFERENCES

1. Gottlieb, B., and Orban, B.: Tissue Changes in Experimental Traumatic Occlusion with Special Reference to Age and Constitution. J. D. Res. 11:505, 1931.
2. Orban, B.: Tissue Changes in Traumatic Occlusion, J.A.D.A. 15:2090, 1928.
3. Glickman, Irving, and Weiss, L. A.: Role of Trauma From Occlusion in Initiation of Periodontal Pocket Formation in Experimental Animals, J. of Periodontology, 26:14-20, Jan. 1955.
4. Box, H. K.: Experimental Traumatogenic Occlusion in Sheep, Oral Health, 25:9, 1935.
5. Sternlicht, Harold C.: Prosthetic Treatment Planning for the Periodontal Patient, Texas Dental Journal, 74-8, 1956.
6. Goldman, H. M., and Cohen, D. Walter: Periodontia, Fourth Edition, St. Louis, C. V. Mosby Co., 1957.
7. Weinman, J.: Progress of Gingival Inflammation Into the Supporting Structures of the Teeth, J. of Periodontology, 12:71, 1941.
8. Collidge, E. D.: Evidence of Excessive Occlusal Stress in Periodontal Tissues of Human Teeth, J. of Dental Research, 15:159, 1935.
9. Orban, B.: Traumatic Occlusion and Gum Inflammation, J. of Periodontology 10:39, 1939.
10. Moyers, R. E.: The Periodontal Membrane in Orthodontics, J. Amer. Dent., A., 40:22, 1950.
11. Moyers, R. E., and Bauer, J. L.: The Periodontal Response to Various Tooth Movements, American Journal of Ortho., 36:572, 1950.
12. Amsterdam, Morton: Discussion of Paper by Reeves, Robert L., The Occlusal Problem in Periodontics, Meeting A.A.P. Atlantic City, N. J., Sept. 28, 1956.
13. Macapanpan, Luz, D., and Weinman, J. P.: The Influence of Injury to the Periodontal Membrane on the Spread of Gingival Inflammation, J. D. Res. 33:263, April 1954.
14. Goldman, H. M., Schluger, Saul, and Fox, Lewis: Periodontal Therapy, St. Louis, The C. V. Mosby Co., 1956, p. 473.
15. Fox, Lewis: The Occlusal Factor in Periodontal Disease, Alpha Omegan, 43:124, 1949.
16. Jankelson, B.: Physiology of the Stomatognathic System, J.A.D.A., 46:375-386, 1953.
17. Jankelson, B.: Physiology of Human Dental Occlusion, J.A.D.A., 50:664, 1955.
18. Leof, M.: Clamping and Grinding Habits; Their Relation to Periodontal Disease, J. American, Dent. A., 31:184, 1944.
19. Nadler, Samuel C.: Bruxism, A. Classification: Critical Review, J.A.D.A., 54:615.
20. Goldman, H. M., Schluger, Saul, and Fox, Lewis: Periodontal Therapy, St. Louis, The C. V. Mosby Co., 1956, p. 45.
21. Gottlieb, B.: Traumatic Occlusion and the Rest Position of the Mandible, Journal of Periodontology, 18:7, 1947.
22. Kraus, Hans: Therapeutic Exercises, Charles C Thomas, Springfield, 1950, p. 276.
23. Ritchey, B., and Orban, B.: The Crest of the Interdental Alveolar Septa, Journal of Periodontology, 24:75-87, 1953.
24. Massler, Maury: Changes in the Lamina Dura During Tooth Movement, A. J. Ortho., 40:364, May, 1954.
25. Morris, Melvin L.: The Diagnosis, Prognosis and Treatment of the Loose Tooth, Oral Surg., Oral Med., and Oral Path., 6:8 and 9, 1953.
26. Stafne, E. C., and Austin, L. T.: A Characteristic Dental Finding in Acrosclerosis and Diffuse Scleroderma, American Journal of Ortho., and Oral Surg. (Oral Surg. Section) 30:25, 1944.
27. Mitchell, David F., and Chaudhry, Arnold P.: Roentgenographic Manifestations of Scleroderma, Oral Surg., Oral Med., and Oral Path., 10:307-309, March 1957.
28. Schluger, Saul: Personal Communications.
29. Schwartz, L. L.: Dental and Facial Pain, J.A.D.A., 51:393-397, Oct., 1955.
30. Hirschfield, Leonard: The Use of Wire and Silk Ligatures, J.A.D.A., 41:647, Dec., 1950.
31. Chaikin, B. S.: Temporary Splinting in Periodontal Therapy, Alpha Omegan, September, 1953.
32. Friedman, N.: Temporary Splinting, an Adjunct in Periodontal Therapy, J. of Periodontology, 24:229, Oct., 1953.

A Study of the Topical Administration of Hydrocortisone Acetate, 9-alpha-Fluorohydrocortisone Acetate and Pyridoxine Hydrochloride Upon Oral Lesions*

by PETER DANTE FERRIGNO, M.S., D.D.S., M.SC., *Columbus, Ohio*

HYDROCORTISONE acetate, 9-alpha-Fluorohydrocortisone acetate, and Pyridoxine hydrochloride have been proposed for treatment of various oral lesions of an inflammatory nature. Literature reports have indicated beneficial results by topical application. However, the need for a controlled study in which application of the various drugs might be used on one side of the mouth and placeboes on the other was indicated.

REVIEW OF THE LITERATURE

Just as there is a characteristic response of the body to systemic stress, referred to as General Adaptation Syndrome (G-A-S), a response to topical stress may elicit a Local Adaptation Syndrome (L-A-S) in which the principle responses are confined to the immediate site of injury. These responses may consist of degeneration, atrophy, necroses, inflammation, hypertrophy or hyperplasia.[1, 2, 3, 4, 5]

Since cortisone, Kendall's compound E, is considered the parent compound from which all of the topically active steroids have been derived and since its systemic activities are so closely related to the topical activities of the other corticoids[6] the author believes that its discussion should be included in this paper. This adrenal cortical steroid seems to act quite specifically upon the intercellular substance of the connective tissue.[4] When cortisone is administered by injection hyaluronic acid is decreased in concentration, altering the chemical composition of the ground substance and increasing its permeability. Cell division is so effected[7] that even when cleavage occurs, the abnormal pattern tends to produce unequal division. This may account for the fibroblasts, which are active in the collagen fibril formation, to be reduced in number and to undergo morphologic changes.[8] Similarly, the vascular permeability is influenced by this hormone.[8] This results in increasing the capillary resistance and reducing edema.[9, 10]

The influx of the polymorphonuclear leukocytes has been shown to be inhibited[11, 12, 13] with a reduction of the total white blood cell count.[10] With the increased vascular tone produced by the cortical hormones, tending to suppress the vascular and cellular inflammatory response to injury[14, 15, 16, 17, 18, 19, 20] there is a marked effect upon wound healing and the formation of granulation tissue.

Because topical application of hydrocortisone acetate and 9-alpha-Fluorohydrocortisone acetate, in the form of ointments have been shown to produce successful results in the treatment of various dermatologic conditions, it was introduced as a treatment for certain oral lesions such as aphthous ulcers, acute and chronic gingivitis, lichen planus, leukoplakia, and many others, with varying results.[21, 22, 23, 24, 25, 26, 27, 28, 29, 30] Beneficial results have been proclaimed with the use of these topical dental ointments in the treatment of aphthous ulcers,[22, 29] acute and chronic gingivitis, including desquamative gingivitis,[21, 23, 26, 29] leukoplakia, glossitis, and burns[29] erosive lichen planus and dilantin gingivitis,[24] angular cheilosis[30] and inflammation associated with immediate dentures.[21] The treatment of psoriasis, lichen planus and sclera derma with these dental ointments were not found to be beneficial.[31, 24, 32]

In a series of experiments of pyridoxine diets on monkeys, certain oral lesions were produced. The systemic administration of this vitamin gave negative results, whereas when it was applied as a topical ointment the lesions disappeared.[33] Some investigators believe that these lesions were produced due to a loss of pyridoxine in the process of the metabolic disturbance.[30] Pyridoxine hydrochloride was applied to, aphthous, ulcers, cheilosis, geographic tongue, and lichen planus with favorable results.[34, 35, 36, 30]

MATERIALS AND METHOD

The dental ointments used in this study for the treatment of the various lesions consisted of:

1. Alflorone Acetate, 0.25% topical ointment (9 - alpha - Fluorohydrocortisone acetate in emollient base;

2. Hydrocortisone Acetate, 2.5% topical ointment with 3.5 mg. Neomycin Base per gram;

3. Pyridoxine Hydrochloride, 2.5% topical ointment.

A series of patients with various oral lesions were supplied with one of the above

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TABLE I

Topical Application of Hydrocortisone

Case No.	*Age*	*Sex*	*Duration of Lesion Before Institution of Treatment*	*Duration of Treatment*	*Diagnosis*
RB-B-01	18	M	3 weeks	3 months	marginal gingivitis
AC-B-02	70	F	3 weeks	2 weeks	chron. atroph. sen. muco.
GC-B-03	49	F	1 month	2 months	herpetic stomatitis
WD-B-04	48	M	3 days	7 days	herpetic stomatitis
WE-B-05	40	M	unknown	2 months	lichen planus
AF-B-06	43	M	unknown	3 months	lichen planus
ML-B-07	29	F	20 years	3 months	periad. muc. nec. rec.
GM-B-08	53	M	5 months	4 months	hyperkeratosis
RN-B-09	19	M	4 days	7 days	prim. herpetic gingivasto.
JP-B-10	62	M	10 years	3 months	desquamative gingivitis
TS-B-11	66	M	9 days	3 months	eros. lich. plan.
AT-B-12	66	F	unknown	2 months	lichen planus
JW-A-13	15	M	1 month	2 weeks	herpetic stomatitis

mentioned topical dental ointments and a placebo for a control. The patients chosen were those that in so far as possible had bilaterally similarly involved areas. The medicinal ointment and its placebo were identified by letters so that the materials would be unknown to the patient. The patients were instructed to apply one-quarter inch of the ointment on one side of the mid-line and a placebo on the other side. The materials were massaged onto the lesions with the index fingers, three times a day (morning, noon and before bedtime). Each patient was instructed to refrain from eating or drinking for one-half hour following the application. No restrictions were imposed on these patients with respect to food selection, smoking, or oral hygiene habits. Periodic observations were made for changes by comparison of photographs taken prior to and during the treatments. Any subjective and objective evidence of improvement was recorded.

EXPERIMENTAL RESULTS

By means of gross observation, 42 cases were studied comparatively and changes between the treated and the controlled side noted. Thirteen cases were treated with Hydrocortisone, 14 with Fluorohydrocortisone, and 15 cases with Pyridoxine.

Table I outlines the cases, duration of treatment and the diagnosis of those treated with Hydrocortisone. No comparative objective difference could be detected throughout any of the series. There was either no change observed between the two sides during and after treatment or similar improvement on both sides.

Subjectively three patients indicated improvement on the side with Hydrocortisone and two patients indicated improvement on the side treated with placebo.

Figures 1 and 2 illustrate a case of primary herpetic gingivostomatitis. A guide to this diagnosis was made in that no improvement was noted following administration of penicillin. The patient was instructed to apply the Hydrocortisone to the upper left arch and the placebo to the right side. Both sides showed a decrease in the intensity of the inflammation in respect to color and tone, so that it was not possible to ascertain whether one side demonstrated any greater beneficial effect than the other (Figure 2).

Figures 3, 4, 5, and 6 illustrate a case of erosive lichen planus treated for three months with Hydrocortisone. Figure 3 shows the lesion at the beginning of the treatment and Figure 4 shows the lesion after two months. There was a tremendous change in this case in that no further signs

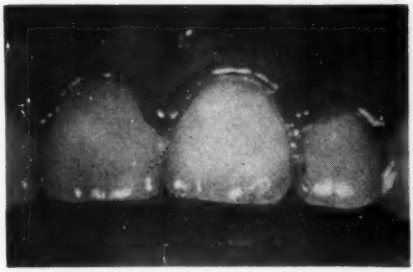

Fig. 1. Primary gingivostomatitis.

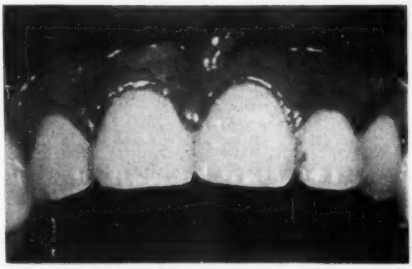

Fig. 2. Primary gingivostomatitis after hydrocortisone.

of erosiveness and erythema could be detected. However, the changes seen between Figures 5 and 6 show similar improvement with the placebo.

Table II outlines those cases treated with Fluorohydrocortisone. Again, in this series, no macroscopic differences were observed. This is illustrated in Figures 7 and 8, in which Figure 7 depicts the appearance of the lesion prior to treatment and Figure 8 shows the changes which occurred in both lesions following treatment. The Fluorohydrocortisone was applied to the right side, the placebo to the left. Figure 9 shows a case of hormonal gingivitis associated with trauma. Fluorohydrocortisone was used on the left half and the placebo on the right half of both the upper and lower arches, no differences could be determined. The lower arch shows the lack of change more clearly since in this area the involvement is more continuous than that shown in the upper arch. In this particular case, there was no

Fig. 3. Erosive lichen planus.

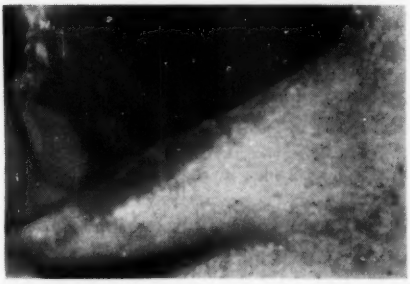

Fig. 4. Same case as Fig. 3 after two months treatment with hydrocortisone.

Fig. 5. Erosive lichen planus.

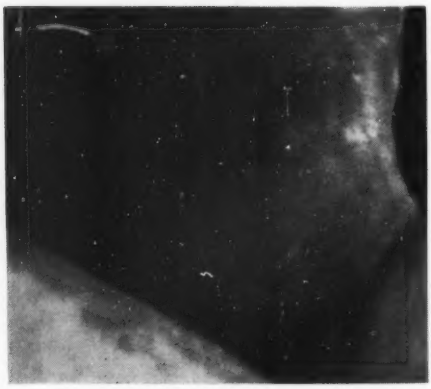

Fig. 6. Same as Fig. 5, treated with the placebo.

improvement throughout the entire two months duration of treatment.

There was only one case in which there were any subjective differences between the two sides. This was a case of periadenitis nucosa necrotica recurrens in which the patient indicated that the controlled side "felt better" than the side on which the medicated ointment had been applied.

Table III outlines the Pyridoxine treated cases. This ointment also produced no objective macroscopic changes. A case of primary herpetic gingivostomatitis was treated for a period of seven days in which the medicated ointment was applied to the left half of the lower arch and the placebo was used on the right side (Figure 10). Daily examinations of the patient showed no differential alterations. In a case of migratory glossitis (Figure 11) in which Pyridoxine was used on the left side of the tongue and a placebo used on the right side, no changes were evident. Also no macroscopic changes were noted in a case of hyperkeratinization of the tongue in which Pyridoxine was used on the right half and the placebo on the left side (Figure 12). No subjective differentiation was noted by any patient in this series.

DISCUSSION

Care was used in the selection of cases, so that in so far as possible, the lesions studied as bilaterally would be similar as possible. In this manner the same individual may serve as both the experimental and control subject. Even this method could have presented certain shortcomings. Although the lesions appeared similar to each other in intensity, size, number or duration, it is impossible to know whether they actually were identical in all respects, microscopically and in their bilogic activity. However, in all cases, the objective symptoms were similar.

TABLE II

Topical Application of Fluorohydrocortisone

Case No.	*Age*	*Sex*	*Duration of Lesion Before Institution of Treatment*	*Duration of Treatment*	*Diagnosis*
NB-A-01	14	F	unknown	1½ months	marginal gingivitis
RB-A-02	18	M	unknown	3 weeks	marginal gingivitis
ME-A-03	74	F	1 year	3½ months	lichen planus
WE-A-04	40	M	unknown	4½ months	lichen planus
AF-A-05	43	M	unknown	3 months	eros. lich. plan.
DH-A-06	23	F	3 months	4 months	lichen planus
PM-A-07	29	F	unknown	1½ months	marginal gingivitis
JO-A-08	25	M	2 days	1 month	periaden. muc. nec. rec.
JP-A-09	62	M	10 years	6 months	desquamative ging.
MS-A-10	40	F	unknown	1½ months	lichen planus
KS-A-11	9	F	6 months	5 months	hormonal ging.
AT-A-12	66	F	unknown	3 months	lichen planus
GW-A-13	25	F	unknown	2 months	migratory glossitis
JW-A-14	15	M	1 month	2 weeks	herpetic stomatitis

Fig. 7. Lesion prior to treatment.

Fig. 8. Same as Fig. 7. Note changes in both lesions (placebo used on left).

The possibility that some of the medicinal agents could have been carried over to the control side by the saliva and the movement of the tongue must be considered. Since it is considered that sufficient absorption of the ointment is accomplished during the time of massaging, one would be led to believe that there should be some difference in the degree of beneficial effects. It is suggested by the author that certain tracing agents should be applied to the medicinal ointment to determine if any is carried to the control side. Moreover, it would be desirable to determine the penetrating ability of the ointment with some tracers to determine whether the medicinal agent reached an area where it could be effective.

The effect of massage has been suggested to explain the equal responses of the controlled and the treated side. In a case, not reported in the experimental results, the author directed a patient with marginal gingivitis to massage Hydrocortisone over one quadrant only and to do nothing to the other. After a two week period, the treated tissue was less erythmatous. Another patient with marginal gingivitis was given a tube of placebo and asked to massage one quadrant and to do nothing to the other side. Again, similar results were obesrved, the side massaged with the placebo showing less erythema.

The claim that the antiphlogistic agents

TABLE III

Topical Application of Pyridoxine

Case No.	*Age*	*Sex*	*Duration of Lesion Before Institution of Treatment*	*Duration of Treatment*	*Diagnosis*
NB-C-01	14	F	unknown	2 months	marginal ging.
RB-C-02	18	M	3 weeks	2 weeks	marginal ging.
AB-C-03	65	F	unknown	4 months	lichen planus
GC-C-04	49	F	1 month	2 months	herpetic stomatitis
WD-C-05	48	M	3 days	7 days	herpetic stomatitis
WE-C-06	40	M	unknown	1 month	lichen planus
AF-C-07	43	M	unknown	3 months	eros. lich. plan.
PM-C-08	29	F	unknown	1 month	marginal ging.
GM-C-09	53	M	3 months	2 months	hyperker. with inflammation
RN-C-10	19	M	4 days	7 days	prim. herpetic ging.
JP-C-11	62	M	10 years	2 months	desquamative ging.
MS-C-12	57	F	4 months	2 months	lichen planus
AT-C-13	65	F	unknown	3 months	lichenoid lesion
AT-C-14	49	F	unknown	2 months	lichen planus
GW-C-15	25	F	unknown	2 months	migratory glossitis

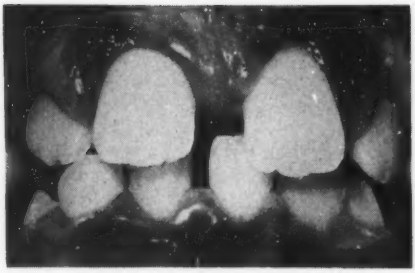

Fig. 9. "Hormonal" gingivitis. Placebo used on right side.

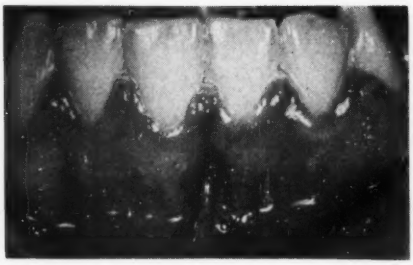

Fig. 10. Primary herpetic gingivostomatitis. Placebo used on right.

could lower the inflammatory response of the involved tissue, making the tissue more susceptible to bacteria or toxins is plausible. In view of this probability if they are used, one of the broad-spectrum antibiotics might be combined with the ointments to limit the possibility of invasion of bacteria. Such ointments are commercially available and are being investigated.

SUMMARY AND CONCLUSIONS

The purpose of this study was to determine the effectiveness of Hydrocortisone Acetate, 9-alpha-Fluorohydrocortisone Acetate and Pyridoxine hydrochloride dental ointments on various oral lesions of an inflammatory nature. This was planned as a pilot study for possible future work with antiphlogistic and associated agents.

Seven cases of marginal gingivitis, one case of chronic atrophic senile mucositis, six cases of herpetic stomatitis, two cases of primary herpetic gingivostomatitis, two cases of periadenitis mucosa necrotica recurrens, 15 cases of lichen planus, three of which were of the erosive type, one case of a lichoid lesion, two cases of hyperkeratosis with inflammation, three cases of desquamative gingivitis, one case of hormonal gingivitis and two of migratory glossitis, were studied. Fourteen were treated with Fluorohydrocortisone, 13 with Hydrocortisone and 15 with Pyridoxine.

The patients were instructed to apply a treatment ointment to the lesions on one side and a control ointment to the lesions on the opposite side, three times daily, for varying lengths of time. The contents of the tubes were not known to the patients.

Kodachrome transparencies were taken at the beginning and throughout the treatment phase at the time of their check-ups.

Macroscopically there were no differences observed between the lesions treated by the drugs and those treated by placeboes.

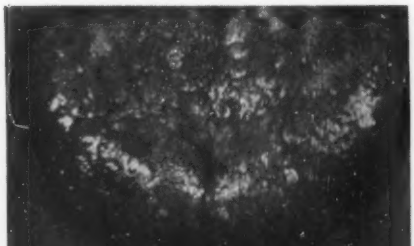

Fig. 11. Migratory glossitis. Placebo used on right.

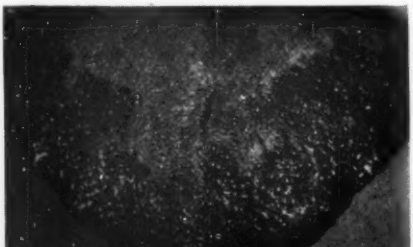

Fig. 12. Hyperkeratinization. Placebo used on left.

On the basis of this controlled study, none of the three ointments used appeared to be beneficial in the treatment of the specific lesions utilized under the conditions of this experiment.

BIBLIOGRAPHY

1. Selye, H.: The Part of Inflammation in the Local Adaptation Syndrome. In: Jasmin, G. and Robert, A., Eds. The Mechanism of Inflammation. Acta. Inc., Montreal, (1953).

2. Selye, H.: Alarm Reaction, The General Adaptation Syndrome, and the Role of Stress and of the Adaptive Hormones in Dental Medicine. Oral Surg., Oral Med. and Oral Path. 7:335, (1954).

3. Selye, H.: Role of Stress and Adaptive Hormones in Dental Medicine. Oral Surg., Oral Med. and Oral Path. 7:255, (1954).

4. Selye, H. and Henser, G.: Fourth Annual Report on Stress. Acta. Inc., Montreal, (1954).

5. Selye, H. and Henser, G.: Fifth Annual Report on Stress. M.D. Publ. Inc., (1955/56).

6. Anonymous: Topical Hydrocortisone. Brit. Med. Jour. 1:530, (1955).

7. Menkin, V.: Effect of Some Steroids and of Corticotrophin (ACTH) on Cellular Activity. Proc. Soc. Exp. Biol. and Med. 82:189, (1953).

8. Bowers, J. Z., Helson, I. H., Bay, R. and Samuels, L. T.: Alterations in 17-hydroxycorticosteroids of Peripheral Blood Following Acute Whole Body Irradiation in the Dog. J. Clin. Endocrinol. and Metab. 12:921, (1952).

9. Arteta, J. L.: Topical Protection Against Hyperargic Inflammation by a Preceding Local Inflammation. Prod. Soc. Exp. Biol. and Med. 82:162, (1954).

10. Moon, V. and Tershakovec, M. A.: The Effects of Cortisone on Blood Cells After Thermal Stress. Proc. Soc. Exp. Biol. and Med. 85:7, (1954).

11. Creditor, M. C., Bevans, M., Mundy, W. L. and Regan, C.: Effect of ACTH on Wound Healing in Humans. Proc. Soc. Exp. Biol. and Med. 74:254, (1950).

12. Dougherty, T. F. and Schneebli, C. L.: Role of Cortisone in the Regulation of Inflammation. Proc. Soc. Exp. Biol. and Med. 75:854, (1950).

13. Germuth, F. G., Ottinger, B. and Oyama, J.: The Influence of Cortisone on the Evolution of Acute Infection and the Development of Immunity. Bull. Johns Hopkins Hosp. 91:22, (1952).

14. Cummings, M. M., Drummond, M. C., Micheal, M., Jr. and Bloom, W. L.: The Influence of Cortisone on Artificially Induced Periotoneal Exudates. Bull. Johns Hopkins Hosp. 90:185, (1952).

15. Ebert, R. H. and Barclay, W. R.: Changes in Connective Tissue Reaction Induced by Cortisone. Ann. Int. Med. 37:506, (1952).

16. Kass, E. H. and Finland, M.: Adrenocortical Hormones in Infection and Immunity. Ann. Rev. Microbiol. 7:361, (1953).

17. Moon, V. H. and Tershakovec, M. A.: Influence of Cortisone Upon Acute Inflammation. Proc. Soc. Exp. Biol. and Med. 79:63, (1952).

18. Moon, V. H. and Tershakovec, M. A.: Effect of Cortisone Upon Local Capillary Permeability. Proc. Soc. Exp. Biol. and Med. 85:600, (1954).

19. Robinson, H. J. and Smith, A. L.: The Effect of Adrenal Cortical Hormones on Experimental Infection. Ann. N.Y. Acad. Sc. 56:757, (1953).

20. Wyann, L. C., Fulton, C. P., Shulman, M. H. and Smith, L. L.: Vasoconstriction in the Cheek Pouch of the Hamster Following Treatment with Cortisone. Am. J. Physiol. 177:335, (1954).

21. Amesbury, R. H.: Hydrocortisone Acetate Dental Ointment for Immediate Dentures. D. Digest. 62:14, (1956).

22. Bergman, S. A.: Topical Application of Hydrocortisone Acetate in The Treatment of Aphthous Ulcers. D. Digest. 60:60, (1954).

23. Duran-Reynals, F. and McCrea, J. F.: The Ground Substance of the Mesenchyme in Inflammation. In: Jasmin, G. and Robert A., Eds. The Mechanism of Inflammation. Acta. Inc., Montreal, (1953).

24. Fisher, A. A.: Topical Use of Hydrocortisone Ointment for Certain Oral Condition. N. Y. State J. Med. 55:2494, (1955).

25. Hoffer, H.: The Treatment of Procaine Dermatitis with Topical Application of Hydrocortisone Dental Ointment. J.C.D.A. 19:383, (1953).

26. Horton, C. P.: Hydrocortisone Acetate in the Treatment of Oral Inflammatory Disease. N. Y. J. Dent. 25:10, (1955).

27. Shroff, F. R.: Steroid Hormones and Periodontal Diseases. Oral Surg., Oral Med. and Oral Path. 7:383, (1954).

28. Spra, M.: Diphenylhydantoin Sodium Hyperplasia: Hydrocortisone Acetate as an Adjunct in its Treatment. Jour. Oral Surg. 13:280, (1955).

29. Strean, L. P. and Horton, C. P.: Hydrocortisone in Dental Practice. D. Digest. 59:8, (1954).

30. Sulke, A. and Yardeni, J.: Hydrocortisone in the Treatment of Oral Diseases Associated with Emotional Stress. D. Digest. 61:448, (1955).

31. Church, R.: Hydrocortisone Ointment in the Eczemas. Brit. Med. Jour. 1:517, (1955).

32. Melton, F. M.: Recent Trends in Dermatologic Therapy. Lancet 1:7, (1955).

33. Robinson, H. B. G.: Personal Communications.

34. Curth, H. O.: Aphtosis; Recurrent Oral and Genital Ulcerations; Involvement of Gastrointestinal Tract. A.M.A. Arch. Dermat. 73:401, (1956).

35. Fisher, A. A.: Aphthosis. A.M.A. Arch. Dermat. 73:401, (1956).

36. Strean, L. P.: Personal Communications.

In Vitro Calcification of Dental Calculus*

by BERNARD H. WASSERMAN, IRWIN D. MANDEL AND BARNET M. LEVY,
New York, New York

DENTAL calculus formation has been studied *in vitro* by a number of investigators.[1,2,3] In their experiments, calculus-like material was produced on extracted teeth by immersing them in saliva or in solutions approximating saliva in composition.

Another approach to the study of calculus formation *in vitro* was suggested by recent work of Mandel, *et al*,[4] in which it was found that when celluloid strips were placed around the lower anterior teeth of patients who form calculus rapidly, calculus was deposited on the strips. The bacterial plaque, which formed on these strips as the first stage in calculus formation, did not begin to calcify for at least three days. Removal of the strips before calcification began in the mouth made available a bacterial plaque which had the potential of becoming calcified.

Numerous investigators studying *in vitro* calcification of bone and cartilage[5] have utilized a standard calcifying solution first suggested by Shipley, Kramer, and Howland.[6] This solution can be modified so that the calcium and phosphorus level and the pH simulate those of saliva.

It appeared to us that immersing celluloid strips with adherent precalcified bacterial plaque into a modified calcifying solution might provide a useful technique for study of calculus calcification *in vitro*. Such a technique is also useful for the study of agents which may be inhibitory to this type of calcification.

MATERIAL AND METHODS

Strips with adherent bacterial plaque were removed from the mouth after 55 to 60 hours and were suspended in a calcifying solution for 4 to 6 days at 37 C. On removal from the solution, the calcified material on the strips was studied by soft X-rays, by staining directly with alizarin red S, and by preparation of histological sections and staining by the von Kossa silver method. As a control, pieces of lip, tongue, mucous membrane and skin were suspended for 6 days in individual flasks containing calcifying solution.

Colonies of *Actinomyces israeli*, originally isolated from bacterial plaque on strips, were cut from blood agar plates and immersed in the calcifying solution for 6 days. In addition, some of the *Actinomyces* colonies were treated with 10% neutral formalin for 15 minutes; samples were then tested for viability, and the remainder were placed in the calcifying solution.

To determine the physico-chemical nature of the *in vitro* calcified material, pooled material from a number of strips was studied by X-ray diffraction techniques using a 19cm. cylindrical camera and both FeK α and CuK α radiation.

Inhibition of calcification was studied in the following manner. Each of the celluloid strips was divided in half. One half was suspended in the usual calcifying solution as a control. The other half was suspended in a calcifying solution to which had been added either Mylase L-1 (Wallerstein), Prolase 100 (Wallerstein), β-glucuronidase, testicular hyaluronidase, or acetylsalicylic acid to make a final concentration of 1 mg. per cc. Acetylsalicylic acid has been used for inhibition of urinary calculus formation:[7] the enzyme preparations have been suggested as inhibitors of salivary calculus formation. In order to determine relative calcification after incubation for 6 days, soft X-rays (5 KVP, 10MA, at 10 sec.) of both the experimental and control portion

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*Supported in part by a grant from the Bristol-Meyers Company, Inc. and in part by a contract between the Office of Naval Research, Department of the Navy, and Columbia University.

of each strip were obtained on the same film.

The calcifying solution used throughout was prepared from a sterilized basal salt solution containing 0.7M NaCl, 0.05M KCl, and 0.22M $NaHCO_3$.[5] This stock solution was diluted tenfold by the addition of sterile water, and K_2HPO_4 was added to make a final concentration of 12 mg% of P. CO_2 was bubbled through the solution to depress the pH to 6.0. $CaCl_2$ was then added to make a final concentration of 4 mg% of Ca. The pH was adjusted to 7.0 by passing compressed air through the solution. The calcium and phosphorus levels were comparable to the ionic concentration of these elements in saliva.

Small (25 and 50 cc.) Erlenmeyer flasks were filled to within ½ inch of their total capacity with calcifying solution. The strips were threaded through a hole in one corner and immersed completely. The other end of the thread was held by a tightly fitting cork stopper which was then sealed with paraffin. The flasks were incubated for 4 to 6 days at 37°C.

RESULTS

When placed in the calcifying solution, celluloid strips with adherent bacterial plaque, showed evidence of calcification by the 4th day. This could be demonstrated by soft X-rays of the strip (Fig. 1), by staining the strip directly with alizarin red S (Fig. 2), and by preparation of histologic sections stained with the von Kossa silver stain (Fig. 3). Most of the strips were kept in the calcifying solution for 6 days, however, to give a heavier deposit. No significant change in pH was evident in any of the solutions at the end of this time.

Pieces of skin, lip, mucous membrane and tongue suspended in the calcifying solution for 6 days did not show evidence of calcification when sectioned and stained with the von Kossa silver stain.

X-ray diffraction study of the calcified material on the strips showed the material to be pure apatite.*

Treatment of the strip with adherent bacterial plaque with 10% neutral formalin for 15 minutes did not affect its ability to calcify, as demonstrated by staining with alizarin red S (Fig. 4). A control portion of the strip with adherent plaque treated with formalin for 15 minutes and then cultured showed that no viable organisms were present. *A. israeli* colonies suspended in the calcifying solution showed considerable calcification as demonstrated by histologic sectioning of the colonies and staining with the von Kossa stain (Fig. 5). Pretreatment with formalin did not affect the ability of the Actinomyces colonies to calcify.

The addition of Mylase L-1 (Wallenstein), Prolase 100 (Wallenstein), β-glucuronidase, and acetylsalicylic acid at a level of 1 mg. per cc. of calcifying solution had little or no effect on the calcification of the strips (Fig. 6). Testicular hyaluronidase at this concentration level had a marked inhibitory effect on the amount of calcification (Fig. 7).

DISCUSSION

Celluloid strips with adherent bacterial plaque material calcify in the modified calcifying solution. The apatite formed is similar to that found in bone, teeth, and salivary calculus formed in the mouth. The failure of lip, skin, tongue and mucous membrane to calcify in this solution indicates that tissues which do not calcify in the body will not calcify *in vitro* under the conditions of the experiment.

Viability of the bacteria does not seem to be essential for calcification of the bacterial plaque or of the Actinomyces colonies. Phosphatase activity may have been unaffected by pre-treatment with formalin. However, the absence of organic phosphates in the calcifying solution precludes

*The X-ray diffraction studies were carried out by Drs. Robert Gorlin and J. W. Gruner at the University of Minnesota.

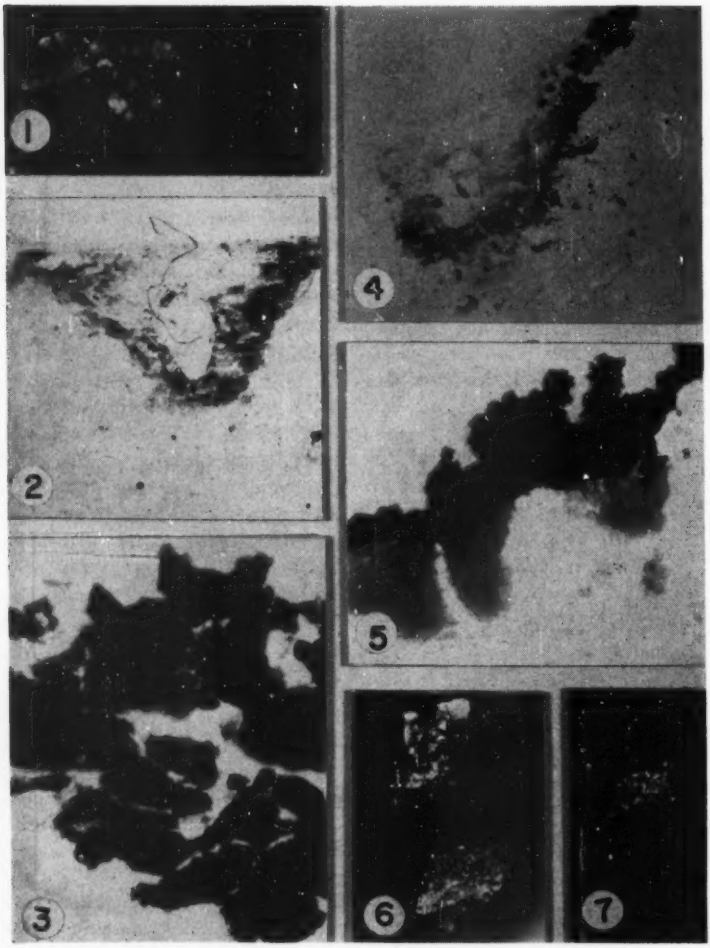


Fig. 1. Soft X-ray of strip with adherent plaque calcified **in vitro.** Magnification 3 x's.

Fig. 2. Strip with adherent plaque calcified **in vitro** and stained with alizarin red S.

Fig. 3. Photomicrograph of section of strip calcified **in vitro** and stained with the von Kossa silver stain (counter-stained with saffranin). Magnification 100 x's.

Fig. 4. Strip with adherent bacterial plaque pretreated with 10% neutral formalin calcified **in vitro** and stained with alizarin red S. Magnification 10 x's.

Fig. 5. Photomicrograph of section of A. israeli colony calcified **in vitro** and stained with the von Kossa silver stain (counter-stained with saffranin). Magnification 100 x's.

Fig. 6. Soft X-ray of control and experimental portions of strip calcified **in vitro** showing the failure of Mylase 1 mg. per c.c. to inhibit calcification. Magnification 3 x's.

Fig. 7. Soft X-ray of control (A) and experimental (B) portions of strip calcified **in vitro** showing inhibition of calcification of B by testicular hyaluronidase 1 mg. per c.c. Magnification 3 x's.

phosphatase activity as part of the *in vitro* calcification mechanism of at least the Actinomyces colonies. There may have been some organic phosphate from saliva present in the bacterial plaque. The bacteria may play a role in salivary calculus formation by virtue of their chemical nature rather than by their enzymatic activity.

Inhibition of calcification by testicular hyaluronidase indicates the presence in bacterial plaque of substances labile to hyaluronidase which have the ability to combine with calcium *in vitro*. Belanger noted a marked inhibition of *in vitro* calcification of demineralized section of tibia by hyaluronidase.[8] It is not possible to identify the chemical substance affected by the hyaluronidase since the spectrum of activity of this type of hyaluronidase is quite broad. In all probability, however, it is a carbohydrate moiety usually found associated with protein. In previous publications,[4,9] the authors pointed out a number of similarities between salivary calculus and other calcifications, especially as regards the presence of carbohydrate-proteins. These carbohydrate-protein complexes may be a part of the "local factor" determining calcifiability of a tissue.[5] Pre-calcified bacterial plaque also shares with rachitic bone, cartilage, atheromatous arteries, etc. the ability to calcify in a similar calcifying solution. This may be additional evidence for a common "local factor" in the mechanism of calcification. Inhibition of calcification by hyaluronidase tends to support the contention that carbohydrate-protein complexes are part of the "local factor."

SUMMARY

1. A technique for studying calcification of pre-calcified bacterial plaque *in vitro* by immersion in a modified calcifying solution is presented.

2. The *in vitro* calcification of the pre-calcified bacterial plaque in a manner similar to other calcifiable tissues, and the inhibition of such calcification by hyaluronidase, may be additional evidence for the presence of a "local factor" common to all calcifiable tissues.

REFERENCES

1. Naeslund, C.: Studies of Tartar Formation. Acta Path. Microb. Scand., 3:637, 1926.

2. Bibby, B. G.: Formation of Salivary Calculus. Dental Cosmos, 77:668, 1935.

3. Prinz, H.: Origin of Salivary Calculus. Dental Cosmos, 63:619, 1921.

4. Mandel, I. D., Levy, B. and Wasserman, B.: Studies on Salivary Calculus. 2. Histochemistry of Calculus Formation. J. of Perio., 28:132, 1957.

5. Sobel, A.: Local Factors in the Mechanism of Calcification. Annals of the N. Y. Acad. of Sci., 60:713, 1955.

6. Shipley, P. G., Kramer, B. and Howland, J.: Studies upon Calcification *In Vitro*. Biochem. J., 20:379, 1926.

7. Prien, E. and Walker, B.: Salicylamide and Acetylsalicylic acid in Recurrent Urolithiasis. J.A. M.A., 160(5):355, 1956.

8. Belanger, L. F.: Discussion of Paper on Mechanism of Calcification. Annals of N. Y. Acad. of Sci., 60:731, 1955.

9. Mandel, I. D. and Levy, B. M.: Studies on Salivary Calculus. 1. Histochemical and Chemical Investigations of Supra and Subgingival Calculus. Oral Surg., Oral Med., and Oral Path. 10:874, 1957.

THE UNIVERSITY OF ARKANSAS

The University of Arkansas has announced the formation of an Institute for Post-Graduate Education in Dentistry. The purpose of the Institute is to promote broadly the advancement of dental education and all related phases of the arts and sciences that contribute directly to the profession.

University officials and the officers of the State Dental Association feel that the Institute will make a significant contribution to the health and welfare of the citizens of Arkansas.

Teaching Dental Anatomy in Correlation with Periodontics

by LLOYD BAUM, D.M.D., M.S., *Loma Linda, California*

IN dental education continuous study has been directed toward the co-ordination of basic sciences and dental technics as they pertain to clinical dentistry. During the past five years in one school,* a definite step has been taken in the co-ordination of the teaching of Dental Anatomy (Tooth Morphology) with that of periodontics. Dental Anatomy, a basic course consisting of lecture and laboratory periods, is taught in the freshman year. Here, for the sake of brevity only the laboratory teaching methods will be discussed.

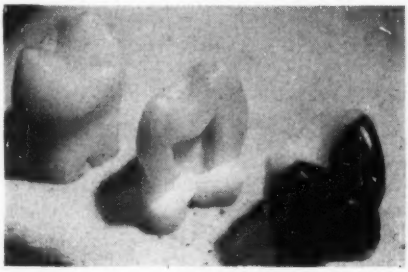


Fig. 1

Most Dental Anatomy laboratory courses are chiefly involved with practice in the carving of individual teeth guided by drawings and specifications in a manual, or models and extracted teeth. These carvings are fabricated of wax, soap, ivorine, and

*School of Dentistry, College of Medical Evangelists, Loma Linda, California.

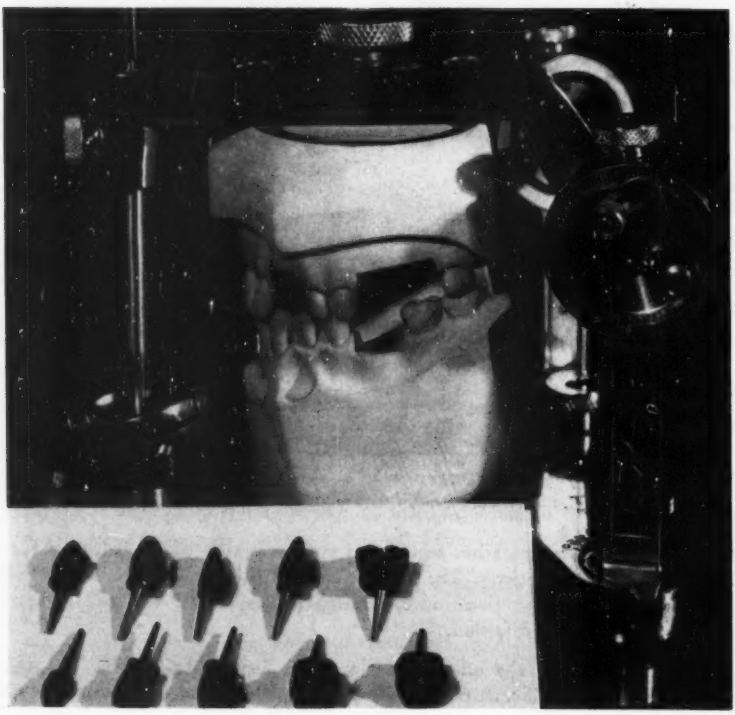


Fig. 2

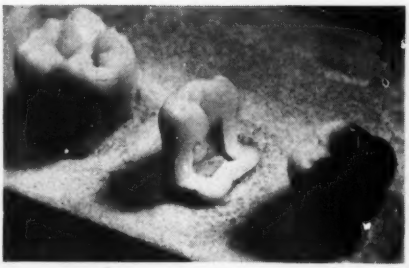


Fig. 3

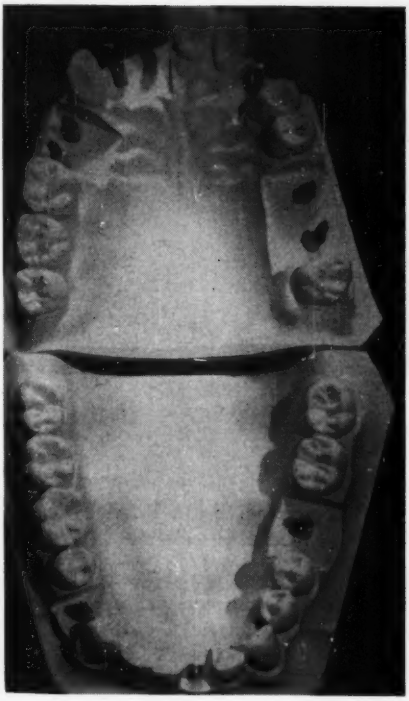


Fig. 4

Fig. 5

other like materials. The predominate objective is to sculpture a tooth from a blank piece of stock material. These may be submitted as individual carved specimens or they may be mounted to form an entire arch of carved teeth.

This laboratory course consists of two phases. The first one, which is a preliminary exercise, gives the student a general introduction to all the permanent teeth. First ten extracted teeth are selected and by use of a Joe Dandy separating disc certain portions of the crowns and roots are removed. Retention for the wax is established with the aid of an inverted cone bur. Inlay wax is used to re-contour the tooth to its original morphology (Figs. 1 and 3).

The remaining portion of the laboratory instruction consists of the following exercise: With a jig these upper and lower casts, with several teeth removed, (See Fig. 2) are mounted in centric occlusion on a Hanau Articulator. These casts have been prepared so that dowel pins are in position for restoring the original teeth. (See Fig. 4) With inlay wax the crowns and coronal portions of the roots are rebuilt to their original contour. They are inspected for proper axial contours, contact points, embrasures, and occlusal contacts. Posterior teeth are carved in centric occlusion with proper occlusal inclined planes. (See Fig. 5) Condyles on the articulator are set and the casts are moved into lateral excursions. Long cusps or steep gliding inclines are properly corrected to harmonize with the existing teeth in the arch. When the teeth are properly "balanced" to protect each tooth from premature contact and undue lateral stress in eccentric positions, the final grooves and detailed anatomy are carved. (See Fig. 6)

Within certain limits teaching should be made as practicable as possible. It is felt that many phases of Dental Anatomy as conventionally taught are good exercises for developing digital skill, for co-ordination the hands and eyes to work together, and for training the student to be accurate in all his movements.

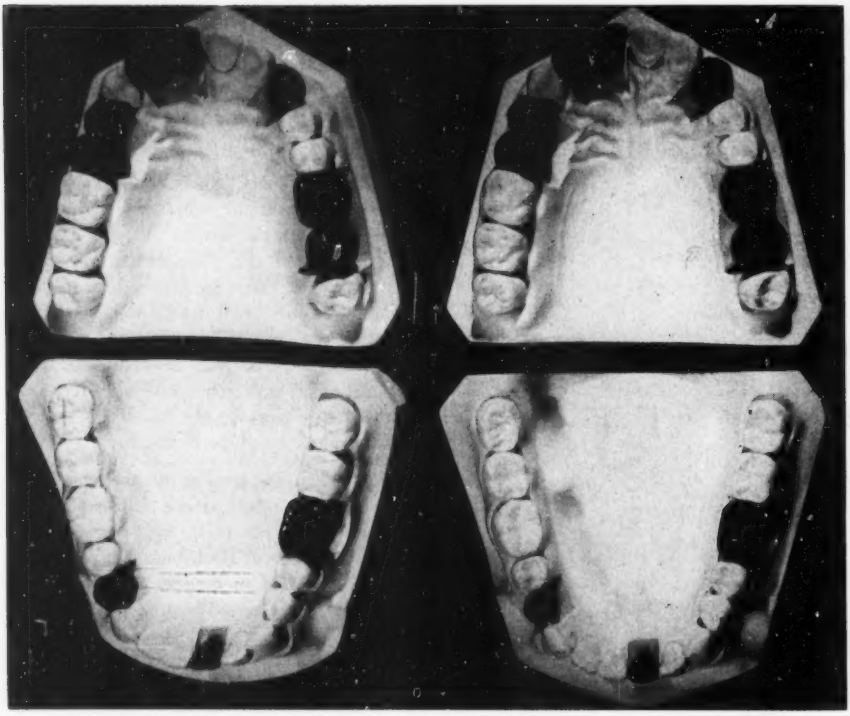


Fig. 6

The use of a material which is non-moldable or which is not repairable is valuable for these exercises. Soap, ivorine, or hard wax are examples of common material which best qualify for this work. However, a practicing dentist is not likely to find all these materials included in his every-day armamentarium. There is little direct practical value derived from carving a tooth out of ivorine. Because the dentist may heat, carve, mold and add to certain portions of the wax pattern as he deems advisable, inlay wax is much more practical for teaching purposes than most other materials.

This approach incorporates another possible advantage from the standpoint of laboratory time. The carving of roots is eliminated. It is felt that the time thus saved can be applied to a better advantage in carving additional crowns.

Perhaps the greatest difference between this and the conventional approach to teaching is physiologic. Probably this is a connecting link between dental technic and applied clinical practice, especially in the field of periodontology. A student must prepare a satisfactory wax carving of a tooth which is esthetically pleasing in form and one which is morphologically correct. More than that he must carve a tooth with a satisfactory buccal curvature to protect the underlying gingival tissue. He must also carve that tooth so that it will have contact in centric occlusion. Moreover, he must carve a tooth which has a broad contact point well toward the buccal to protect the interproximal gingival tissue and pre-

vent the formation of a "food trap." The marginal ridges on that tooth must be well formed and correctly placed in relationship to the marginal ridges on the adjacent teeth. With proper emphasis the foundation for correction of occlusal disharmonies can be embedded in the minds of the student as early as the freshman year.

These fundamentals have their direct clinical application but this is not all. The beginning student should be given the proper orientation to balanced occlusion. He must be impressed that the mouth does not contain individual teeth but series of teeth forming arches which must function as an integral unit. Even as early as the freshman year he should be informed of "supporting cusps," and their importance to occlusion. He should be able to recognize and to relieve simple cases of cuspal interference on the articulator, especially as it might apply to oral health and the protection of the supporting tissues.

In spite of the fact that this type of introduction to dentistry in one of the first technic courses has borne fruit in clinical years, additional time may be necessary before a fair evaluation of this teaching approach will be possible.

TUFTS UNIVERSITY — SCHOOL OF DENTAL MEDICINE THE NINTH ANNUAL BERKSHIRE CONFERENCE IN ORAL PATHOLOGY AND PERIODONTOLOGY

The Division of Graduate and Postgraduate Studies of Tufts University School of Dental Medicine will conduct the Ninth Annual Berkshire Conference in Oral Pathology and Periodontology at Eastover in Lenox, Mass., June 15-19, 1958. The conference will consist of four days of lectures, seminars and panel discussion regarding the clinical problems by outstanding teachers and clinicians in dentistry, medicine and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities with afternoons open for recreation. This is a conference for general practitioners as well as those interested in a specialized practice. The central theme of the conference will be "Gerodontics—Oral Problems Associated with Aging."

Included amongst the faculty are Dr. Joseph L. Bernier, Armed Forces Institute of Pathology, Washington, D. C.; Dr. Irving Glickman, Tufts University School of Dental Medicine, Boston, Mass.; Dr. Max Gratzinger, Chicago, Illinois; Dr. Bernard Jankelson, University of Washington School of Dentistry, Seattle, Washington; Dr. Herbert Mescon, Boston University School of Medicine, Boston, Mass., Dr. Edward J. Steiglitz, Washington, D. C., and Dr. Joseph P. Weinmann, University of Illinois Dental School, Chicago, Illinois.

For further information and application write to Ninth Annual Berkshire Conference, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Massachusetts.

American Academy of Periodontology
President's Address—1957

by STANLEY C. BAKER, *Greenwood, South Carolina*

MEMBERS of the Academy of Periodontology: I cordially welcome you to your 43rd Annual meeting. I appreciate the honor and privilege that you have bestowed upon me as President of your organization and I will try to make this address short, business-like and to the point.

From year to year, the demands of the Journal of Periodontology have increased to the place that it is impossible for the editor, Dr. Maynard K. Hine to carry on without additional help. He has rendered a wonderful service as our editor and has always executed his duties most capably, but the time has come, I believe, when it is necessary to give him more clerical aid. You are aware, I am sure, that Dr. Hine is a tireless worker, but in view of the enormous amount of time and effort he expends on our magazine, I would like to recommend that the Academy provide him with whatever clerical and stenographic aid he feels that he needs to lighten his duties as editor of our publication.

The project which, in my opinion, is of utmost importance at this time is a "Workshop" for teachers and graduates in Periodontia. This idea is not new for it has been suggested by several former presidents and was forcibly brought before us two years ago by Dr. Harold Ray. Among our membership in the Academy are teachers in the field of Periodontia from practically every dental college in this country. Who can better work out the problems of improving our dental curricula, both graduate and undergraduate, and the standardizing of our nomenclature than the men who guide the students through their training period? I would like to see this "Workshop" held annually. Dr. Ray has, through untiring effort, secured funds toward defraying the expenses of our first meeting which we plan to have in San Francisco in 1958. This has already been set up and, with Academy support, can be successfully carried out. Some members, with whom I have discussed this matter, feel that a more centrally located city for the meeting place might attract more teachers, and that, since the "Workshop" would be primarily for teachers, perhaps a Summer meeting would be more satisfactory. Others feel that perhaps we could combine the Spring Seminar at Ann Arbor with the "Workshop"—which might prove a happy solution. The time and place can be discussed and decided upon later. My feeling is that this "Workshop" will be a decisive step forward in the field of periodontia. It is my fervent belief that many problems can be solved more easily through the medium of a "Workshop," thus giving mutual advantage to both teachers and periodontists through the exchange of ideas and practical experience. Standardization of our nomenclature in *all* colleges would more than repay us for the time and money spent to get teachers to attend the "Workshop." This association of teachers and periodontists would also make it easier for the American Board Examiners to phrase their examination questions and would qualify all applicants on an equal basis. Since I feel so strongly on the need of an *Annual* "Workshop," I am going to recommend to the Academy that we support such a program—the place, the time, and the program to be determined later.

Another problem which has worried me considerably this past year, and which faces the Academy at this time, is the office of the Secretary. The Academy has grown, in 43 years, from a small group to a large organization with the result that the duties of our Secretary have grown to mammoth proportions. I've had the pleasure this year of visiting Dr. Chamberlain in Peoria, and I discovered that the facilities are inadequate to handle all of the duties that are his. Dr. Chamberlain has done an outstanding job, but as I see

it, he needs more space than his dental office affords as well as additional equipment such as extra files, mechanical aids, etc. to operate an efficient, up-to-date Home Office befitting an organization such as ours. As I have said before, Dr. Chamberlain has done a masterful job but he is working under extreme difficulties. He has given much valuable time from his dental practice—very willingly, it is true—but I would certainly like to see some of the physical effort lightened. Mrs. Hill deserves commendation also, and we appreciate her conscientious effort, for she too, has been working under difficulties due to the distance she has to travel. I therefore recommend that this body go on record as giving the Council the authorization to set up a Home Office for the Secretary of the Academy, allowing him to rent special office space, to purchase necessary equipment and furnishings and to employ, in addition to Mrs. Hill, such stenographical and clerical aid as will facilitate the conducting of Academy affairs, thus minimizing Dr. Chamberlain's time away from his dental practice. Our valuable records are soon to be moved from Birmingham, Michigan, to Peoria and a safe or a vault should be purchased to house them for they are priceless possessions and should harm come to them, they could never be replaced because there are no duplicates available. I urge you to act on this recommendation immediately.

Next, I'd like to reiterate Dr. Kesel's recommendation of last year that more dental information be disseminated through the medium of radio and television. Commercials are often misleading and while viewers are interested in the programs they are viewing, pertinent dental facts may be substituted for the commercials if they are properly brought to the attention of the advertisers.

The By-Laws Committee has brought in recommendations for changes which are very apropos and should be adopted. I recommend that this be done.

Dr. James E. Aiguier also deserves a word of praise. We are indeed fortunate to have him as our able Treasurer. I have been particularly impressed with his business executiveship in handling the finances of the Academy.

The Scientific Sessions Committee is very important, in fact, most important. These men are carefully selected for their foresight and good judgment in outlining the two-year program of the Academy meetings and in choosing and assisting a program chairman. My thanks to this Committee and to all Committees and Committeemen for their excellent cooperation. It is all reflected in the wonderful program arranged for us this week-end.

Last, but not least, I wish to thank the Executive Council for their forbearance and for their kind guidance through this year. Dr. Kesel, as Chairman of Council, has my utmost admiration for the way that he has conducted our meetings.

Credit is due to the whole Academy membership for interest and support, which I appreciate. And I'd like to extend here a welcome to all visitors with us today.

Report of the Committee on the President's Address

President Stanley C. Baker presented his presidential address before the general assembly of the American Academy of Periodontology on Thursday morning, October 31st, 1957, and expressed his appreciation for the honor and privilege bestowed upon him to serve as president of this organization for the past year. As we all know, the President experienced a strenuous and busy, yet smooth and cooperative year, executing the myriad number of details on which decisions must be accomplished at his office level.

Dr. Baker thanked the officers and members of various committees and to the Executive Council for their excellent cooperation, forbearance and guidance throughout the year. In addition, the President commented that the success of this organization is due primarily to the whole academy membership, for their support and interest.

The recommendations which were made by the President in his address may be summarized as follows:

1. Increased clerical aid for our Journal Editor, Dr. Maynard K. Hine.

2. Academy support for the initial "Workshop" for teachers in periodontology and suggested it be an annual event.

3. That suitable separate and additional space be made available for a home office in Peoria, with the necessary equipment, furnishings, and clerical help to aid Dr. Chamberlain in executing the increased duties of his office as Secretary of the Academy.

4. That the Academy pursue the recommendation presented last year by Dr. Robert Kesel—that dental education be promoted and brought before the general public, by presenting interesting dental health facts on radio and television. The Committee on Public and Professional Relations was formed and is presently actively engaged in planning projects for educating the general public in preventive dentistry.

We are happy to report that the Executive Council has acted favorably on all these four recommendations and they have been read into the minutes of their meeting. This reflects both the excellent leadership and good judgment of our President.

It is with sincere and warm affection that we express our appreciation to Dr. Stanley Baker for the guidance, foresight and vision which he has executed with dignity and dispatch in bestowing upon us the many rewarding benefits of his counsel.

Dr. Raymond E. Johnson
Dr. Robert L. Dement
Dr. E. A. Archer, Chairman

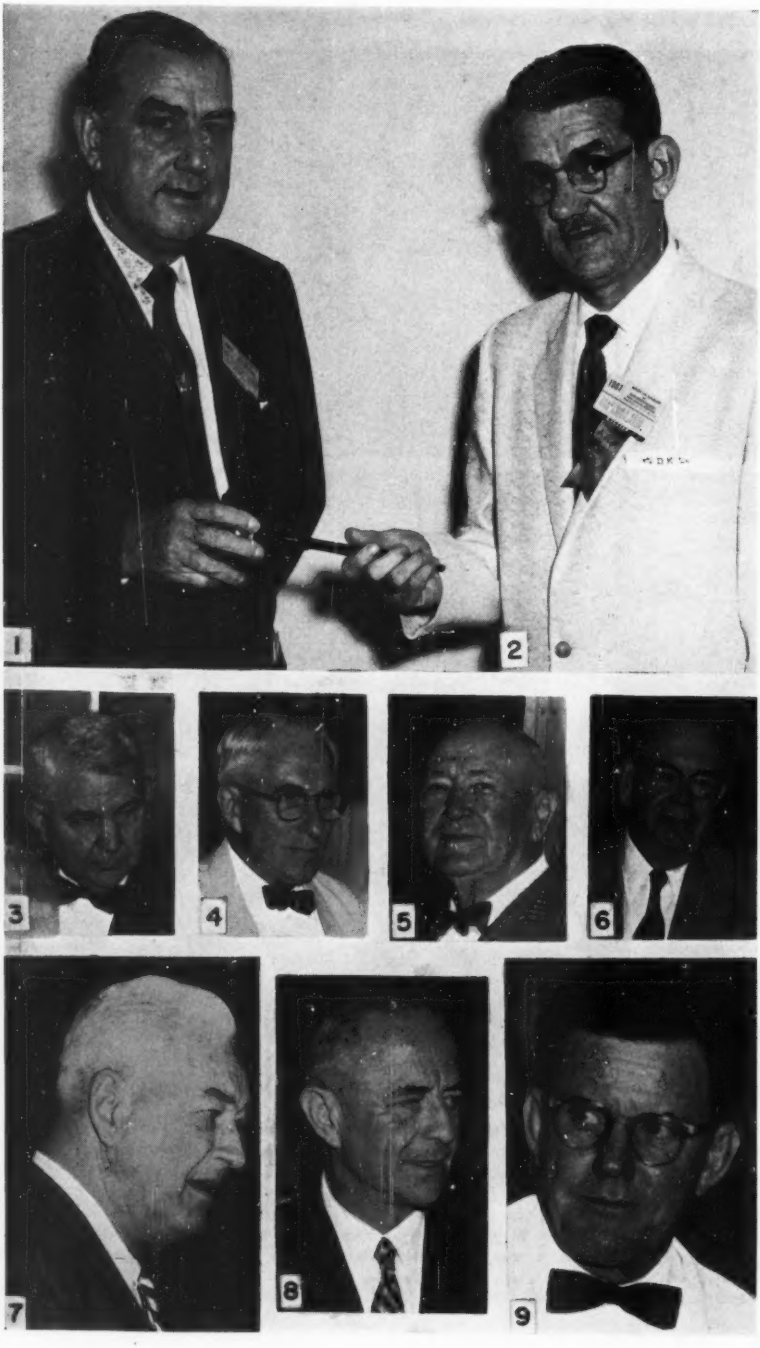
UNIVERSITY OF IOWA

A one day symposium on Newer Drugs and Their Dental Application will be sponsored by the College of Dentistry and the Iowa Center for Continuation Study June 14, 1958. This conference will include discussions of The Tranquilizing Drugs by Dr. Hugh H. Keasling; The Antibiotic Drugs by Dr. V. S. Hinds; A Short History of Dental Therapeutics by Dr. James H. Wick; Prescription Writing, Storage and Dispensing of Drugs by Dr. Seymour M. Blaug; Drugs Used in the Modern Practice of Endodontics, Dr. Arne M. Bjorndal, and Drugs Used as an Adjunct to Periodontal Therapy by Dr. Richard E. Bradley.

For further information write Dr. William D. Coder, Coordinator of Conferences, State University of Iowa, Iowa.

Informal pictures taken at the 1957 Annual Meeting of the American Academy of Periodontology which was held in Miami. Photographs by Dr. Howard Hartman of Cleveland.

(1) Arnold Ariaudo, San Diego, California; (2) Evert A. Archer, Chicago, Illinois; (3) Perry Ratcliff, San Bernardino, California; (4) D. P. Nachazel, Milwaukee, Wisconsin; (5) R. G. Kesel, Chicago, Illinois; (7) H. Roger Turner, Miami, Florida, and friend; (8) Samuel Parks, Dallas, Texas; (9) Mrs. Maynard K. Hine, Indianapolis, Indiana; (10) Maynard K. Hine, Indianapolis, Indiana; (11) Carleton Williams, San Diego, California; (12) James Aiguier, Philadelphia, Pennsylvania; (13) Gordon Agnew, San Francisco, California; (14) Clark E. Chamberlain, Peoria, Illinois.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OFFICERS — 1958

AMERICAN ACADEMY OF PERIODONTOLOGY

Donald A. Kerr ..... President
Joseph L. Bernier ..... President-Elect
Clarke E. Chamberlain ..... Secretary
James E. Aiguier ..... Treasurer

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Stanley C. Baker ..... Chairman
Donald A. Kerr ..... President
Joseph L. Bernier ..... President-Elect
Clarke E. Chamberlain ..... Secretary
James E. Aiguier ..... Treasurer
Craft A. Hopper 1958
Charles H. M. Williams 1958
James P. Hollers 1959
Frank E. Beube 1959
Evert A. Archer 1960
Richard Chace 1960

AMERICAN BOARD OF PERIODONTOLOGY

Harold J. Leonard, Sec'y.-Treas. 1958
Harry Lyons 1958
B. O. A. Thomas 1959
Henry Goldman 1959
Clarke E. Chamberlain 1960
Raymond E. Johnson 1960

JOURNAL OF PERIODONTOLOGY

Maynard K. Hine ..... Editor

SPECIAL COMMITTEES — 1958

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Edward B. Spalding, Samuel R. Parks } Co-Chairmen
Raymond E. Johnson
James A. Aiguier
Donald A. Kerr

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Harold G. Ray, Chairman
Willard Fleming
Maynard K. Hine
James E. Aiguier
Donald A. Kerr
Robert Reeves

On opposite page . . . . . ←

Retiring President Stanley C. Baker (1) hands the badge of office to the new President of the American Academy of Periodontology, Donald A. Kerr (2).

(3) Samuel Parks, Dallas, Texas; (4) Harold J. Leonard, New York, New York; (5) Olin Kirkland, Montgomery, Alabama; (6) Craft Hopper, Paterson, New Jersey; (7) Allison James, Beverly Hills, California; (8) Dickson G. Bell, San Francisco, California; (9) John S. McKenzie, Miami, Florida.

STANDING COMMITTEES OF GENERAL ASSEMBLY — 1958

	Term Expires:
CONSTITUTION and BY-LAWS	
James P. Hollers, Chairman	1958
Floyd E. Lytle	1959
Perry A. Ratcliff	1960
BUDGET and AUDIT	
Joseph L. Bernier, Chairman	
Clarke E. Chamberlain	
James E. Aiguier	
NOMENCLATURE and CLASSIFICATION	
Henry Goldman, Chairman	1960
Joseph L. Bernier	1958
Harry Lyons	1959
EDUCATION	
Frank E. Beube, Chairman	1958
C. Kenneth Collings	1959
Henry M. Swenson	1960
RESEARCH IN PERIODONTOLOGY	
Sigurd Ramfjord, Chairman	1958
Harold G. Ray	1959
Irving Glickman	1960
SCIENTIFIC SESSIONS	
D. P. Nachazel, Chairman	1959
Evert A. Archer	1958
B. O. A. Thomas	1960
ETHICS	
Mildred W. Dickerson, Chairman	1958
John S. McKenzie	1959
Bernard S. Chaikin	1960

AD INTERIM
- Stanley C. Baker, Chairman
- Donald A. Kerr
- Joseph L. Bernier
- Clarke E. Chamberlain
- James E. Aiguier
- B. O. A. Thomas
- James P. Hollers

MEMBERSHIP	
Hunter S. Allen, Chairman	1958
Robert Dement, Co-Chairman	1959
Dickson G. Bell	1958
Robert G. Kesel	1960
M. Monte Bettman	1960

PUBLICATIONS
- Maynard K. Hine, Chairman
- Edgar D. Coolidge
- Balint Orban
- B. O. A. Thomas

SUB-COMMITTEE
JOURNAL SUBSCRIPTIONS
- G. P. Ivancie, Chairman

PUBLIC and PROFESSIONAL RELATIONS
- Saul Levy, Chairman
- G. P. Ivancie
- Perry Ratcliff
- Francis Fabrizio
- Abram Cohen
- Robert Kesel

SUB-COMMITTEES OF SCIENTIFIC SESSIONS — 1958

(Arrangements for Annual Meeting)

LOCAL ARRANGEMENTS
- Samuel R. Parks, Chairman
- James P. Hollers
- C. Kenneth Collings
- Walter Stout
- John Swindle, Jr.

PROGRAM
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- John Prichard
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- Nancy J. Holmes
- Joseph Reed
- Richard Bryarly

ROUND-TABLE LUNCHEON
- Justin D. Towner, Co-Chairman
- James P. Hollers, Co-Chairman

SPRING CONFERENCE
- James Robinson, Chairman '58
- Mark Gilson, Chairman '59
- Romle Romine

AMERICAN ACADEMY OF PERIODONTOLOGY SPONSORS WORKSHOP FOR TEACHERS OF PERIODONTOLOGY

Plans are now almost completed for the workshop for teachers of periodontology which is being sponsored by the American Academy of Periodontology. This workshop will be held at the University of California School of Dentistry in San Francisco June 23, 24 and 25, 1958. At least one teacher from every dental school will be in attendance to discuss various problems associated with the teaching of periodontology. The outline of the program was published in the January issue of the Journal. Because of the nature of the program, attendance is definitely limited to teachers.

The expenses for this program are being underwritten by the Procter & Gamble Company, Lactona, Inc., Fund for Dental Education, Inc., University of California and the American Academy of Periodontology.

PERIODONTOLOGY TODAY

Published by Southwest Society of Periodontists

The Southwest Society of Periodontists has begun publication of an interesting and informative bulletin which they have entitled "Periodontology Today." It is their plan to issue this quarterly and at present it is in the form of a Newsletter. The bulletin includes announcements of meetings and a series of abstracts from journals which not all periodontists and practitioners read. The editor of this publication is Dr. Cliff Ochsenbein and he is assisted by co-editors Latimer and Peck.

We wish to congratulate the Southwest Society of Periodontists on this publication.

PRIZE DR. H. C. RENE JACCARD

The "Prize Dr. H. C. Rene Jaccard" will be presented for the second time during the XV Congress of the "ARPA Internationale" in Paris, July 7 to 12, 1958. The prize will be presented for the best original scientific work on periodontal disease published or performed between 1956 and 1958. It is not necessary for the author to be a member of the ARPA.

The six copies of the papers should be sent, by registered mail, to the president of the ARPA Internationale (Prof. A. J. Held, Institut de Medicine Dentaire, 30 rue Lombard, Geneva, Switzerland) before May 15, 1958.

UNIVERSITY OF PENNSYLVANIA SCHOOL OF DENTISTRY

Applications are requested for a Fellow in Oral Pathology supported by the American Cancer Society, tenable at the Department of Pathology, School of Dentistry, University of Pennsylvania, for a period of one year beginning August 1, 1958. A stipend of $3600 accompanies the fellowship.

Each candidate should submit a curriculum vitae and letters of reference. Reply to Dr. C. E. Wilde, Jr., Department of Pathology, School of Dentistry, University of Pennsylvania.

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES TWELFTH ANNUAL MEETING

The American Academy of Dental Medicine will hold its Twelfth Annual Meeting in Montreal, Canada, at the Sheraton Mount Royal Hotel, on Thursday, Friday and Saturday, May 29, 30 and 31, 1958.

An impressive scientific program has been arranged for this three-day seminar, presenting the interrelationship of oral medicine and general medicine, and highlighting such topics as the dentist and the endocrine problem, the internist, the obstetrician, the pharmacologist, inflammatory diseases of the mucous membranes of the mouth, contact dermatitis of the face and hands. Participating are: Dr. L. P. Ereaux, Department of Medicine of McGill University; Dr. Allen Gold, Consultant in Endocrinology, Queen Mary Veterans' Hospital; Dr. G. W. Halpenny, Department of Medicine, Queen Mary Veterans' Hospital; Dr. Robert F. Harvey, Faculty of Dentistry, McGill University; Dr. Georges Leclerc, Department of Medicine, University of Montreal; Dr. J. L. Macarthur, Montreal General Hospital; Dr. Kenneth I. Melville, Department of Pharmacology at McGill University.

A special feature of the program will be a presentation by the Cleft Palate Team of Montreal Children's Hospital, under the Chairmanship of Dr. F. M. Woolhouse, who will discuss the overall treatment and rehabilitation of the cleft palate patient.

Honorary Membership in the Academy will be accorded Dr. Adelard Groulx and Dr. Don Gullett.

All members and other interested physicians and dentists are cordially invited. A program of entertainment and sightseeing has been arranged. For programs and reservations address: Dr. George Witkin, 45 So. Broadway, Yonkers 2, New York.

DENTAL MUSICIANS NEEDED

Dentists and members of allied professions are invited to become members of two doctors' orchestras in New York City. Non-medical musicians may also join. The Doctors' Orchestral Society of New York rehearses Thursday evenings in the auditorium of the Stuyvesant High School, 15th Street and First Avenue, New York City at 8:30 P. M. The Brooklyn Doctors' Symphony meets Wednesday evenings in the auditorium of the Brooklyn High School of Home Making at 901 Classon Avenue, Brooklyn at the same time. Both groups are rehearsing now.

The Brooklyn group presented its first concert of the 1957-58 season on Friday, February 28, 1958 at 8:30 P. M. at the Brooklyn High School of Home Making.

For further details contact, Dr. Benjamin A. Rosenberg, 909 President Street, Brooklyn 15, New York.